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LIST OF SECTIONS.

CONTENTS OF VOLUME II.

SECTION		PAGE
50.	PHTHISIS,	399
51.	RESPIRATORY NEUROSES,	566
52.	ASTHMA,	566
53.	WHOOPING-COUGH—PERTUSSIS,	600
54.	HYSTERICAL NEUROSES,	616
55.	PERIODIC RESPIRATION,	617
56.	CHEYNE-STOKES BREATHING,	617
57.	AFFECTIONS OF THE PLEURA,	625
58.	CLASSIFICATION OF THE AFFECTIONS OF THE PLEURA,	637
59.	THE ACUTE INFLAMMATORY PLEURISIES,	640
60.	DRY PLEURISY—PLEURITIS SICCA,	653
61.	PLEURITIS EXUDATIVA,	658
62.	SERO-FIBRINOUS EFFUSION,	669
63.	PURULENT EFFUSION—EMPYEMA,	699
64.	TUBERCULAR PLEURISY,	746
65.	DIAPHRAGMATIC PLEURISY,	750
66.	DOUBLE PLEURISY,	753
67.	SYMPHYSIS PLEURÆ (Pleuritic Adhesion),	753
68.	HYDROTHORAX, HYDROPS PLEURÆ, DROPSY OF THE PLEURA,	756
69.	HÆMORRHAGIC EFFUSION,	759

SECTION		PAGE
70.	HÆMOTHORAX,	761
71.	CHYLOTHORAX,	765
72.	PNEUMOTHORAX,	767
73.	AFFECTIONS OF THE DIAPHRAGM,	827
74.	DIAPHRAGMATIC HERNIA,	831
75.	HERNIA OF THE LUNG,	835
76.	SYPHILIS OF THE LUNGS AND PLEURA,	836
77.	PNEUMONO-MYCOSES,	840
78.	ACTINOMYCOSIS OF THE LUNG AND PLEURA,	841
79.	ASPERGILLOSIS,	856
80.	COCCIDIOSIS — PROTOZOAN (or COCCIDIOIDAL) INFECTION OF THE LUNG,	859
81.	BILHARZIA HÆMATOBIA IN THE LUNG,	860
82.	HYDATIDS OF THE LUNG AND PLEURA,	860
83.	NEW-GROWTHS OF THE LUNGS AND PLEURA,	872

A General Index to the work will be found at the end of this Volume.

The Sections dealt with in Volume I. are as follows:—

CONTENTS OF VOLUME I.

SECTION		PAGE
1.	Introductory,	1
2.	Malformation of the Respiratory Organs,	18
3.	General Classification of Diseases of the Respiratory Organs,	19
4.	Diseases of the Air-Passages,	19
5.	Respiratory Obstruction,	19
6.	Foreign Bodies,	24
7.	Catarrhal Laryngitis,	31
8.	Membranous Laryngitis,	38
9.	Œdema of the Larynx (Œdema Glottidis),	53
10.	Nervous Affections of the Larynx Producing Respiratory Orstruction,	56
11.	Congenital Infantile Stridor,	62
12.	Abductor Paralysis,	65
13.	Perichondritis, Necrosis, and Caries of Cartilage,	70
14.	Laryngitis Tuberculosa—Laryngeal Phthisis,	74
15.	Syphilis of the Air-Tubes,	84
16.	Cicatricial Stricture of the Air-Tubes,	87
17.	Tumours in the Air-Tubes,	91
18.	Obstruction to the Air-Tubes from Without,	97
19.	Bronchitis,	108
20.	Acute Bronchitis of the Larger Tubes—Tracheo-Bronchitis Acuta,	147
21.	Capillary Bronchitis—Acute Bronchitis of the Smaller Tubes—Bronchiolitis—Suffocative Catarrh,	150
22.	Acute Bronchitis of the Aged—Asthenic Bronchitis—Peripneumonia Notha,	155
23.	Chronic Bronchitis,	156
24.	Special Forms of Chronic Bronchitis,	159
25.	Bronchitis Putrida, Fetida, Septica,	163
26.	Plastic Bronchitis—Fibrinous or Membranous Bronchitis— Bronchial Croup—Bronchitis Crouposa—Bronchial Polypus,	167
27.	Secondary Bronchitis,	179
28.	Bronchiectasis,	184
29.	Pneumo-Koniosis—Inhalation Diseases of the Lung,	192

SECTION		PAGE
30.	Ordinary, Genuine, True, Large-Lunged Emphysema,	200
31.	Senile Emphysema—Small-Lunged Emphysema—Senile Atrophy of the Lung—Atrophous Emphysema,	218
32.	Hypertrophy of the Lung—Complementary, Compensatory, or Vicarious Emphysema—Relaxed Lung—Paralysis of the Lung,	220
33.	Interstitial or Interlobular Emphysema,	223
34.	Atelectasis—Collapse—Apneumatosis,	225
35.	Hyperæmia—Congestion—Hypostatic Congestion and Œdema,	231
36.	Œdema—Serous Infiltration (Laennec)—Transudation—Inundation (Rokitansky),	242
37.	Acute Pneumonia, Croupous Pneumonia, Lobar Pneumonia, Pleuro-Pneumonia,	245
38	Metastatic Pneumonia—Septic, Embolic, Hæmatogenous Pneumonia,	312
39.	Interstitial Pneumonia,	314
40.	Chronic Pneumonia,	320
41.	Broncho-Pneumonia—Lobular, Disseminated, Patchy Pneumonia,	323
42.	The Broncho-Pneumonia of Little Children,	326
43.	Pulmonary Infarct—Pulmonary Apoplexy—Embolism and Thrombosis,	350
44.	Pulmonary Infarct,	351
45.	Pulmonary Embolism,	354
46.	Pulmonary Thrombosis,	361
47.	Abscess of the Lung,	362
48.	Gangrene of the Lung,	366
49.	Hæmoptysis,	375

DISEASES OF THE ORGANS OF RESPIRATION.

50. PHTHISIS.

HISTORY.

Phthisis was a term employed by ancient writers to indicate what we should call a wasting disease. It was soon associated with the presence of destructive lesions in the lung, lesions which were described as suppuration, ulceration, and abscess, and which were not regarded as differing from the similar processes in other parts of the body. Still Hippocrates was familiar with the clinical symptoms of consumption and gave a good description of the disease.

Tubercle, in the same way, was used simply as a descriptive anatomical term to designate any small morbid mass irrespective of its nature, and it was without special significance.

When wasting was found to be associated with similar lesions in other parts of the body besides the lung, phthisis was still the word employed, and thus arose the terms phthisis pulmonum, phthisis renum, phthisis intestinalis, etc.

This continued to be the state of knowledge even in Galen's time. It was not until the end of the sixteenth century that phthisis of the lung came to be regarded as a special form of disease. It was then that the peculiar masses found in the lung in phthisis were recognised as standing in close relation to the destructive lesion, and from this time the term tubercle came to have a special meaning attached to it.

Morton, writing in 1689, distinguished various forms of phthisis, but referred their origin in all cases alike to these tubercles, and thus laid the foundation of the subsequent theories of the disease. Shortly after, in 1700, Manget described similar tubercles in other organs; but all these observations were forgotten for a hundred years, until at the end of the eighteenth century Baillie's writings raised anew the question of the true nature of phthisis.

Baillie described tubercles as of common occurrence in phthisis and as leading to suppuration, but he distinguished the nodular from the diffuse lesions, and regarded the latter as distinct from tubercle and of a different nature. He called it scrofulous material on account of its resemblance to the enlarged lymphatic glands commonly called scrofulous, and he invented the term caseous or cheesy to describe its appearance. This distinction, however, was not long maintained, for Portal, writing soon after, used the terms tubercular and caseous as synonymous.

Bayle, in 1810, was the first to clearly recognise the connection between the phthisis and tubercular processes in other organs, and to stamp tuberculosis as a general disease. He referred its origin to a peculiar diathesis which he called the tubercular or scrofulous. Phthisis he regarded as a specific affection associated with inflammation, catarrh, and hæmoptysis, but not caused by them.

He described also small granulations of cartilaginous nature and consistence, of opaque appearance, and without a tendency to soften, and he named the cases in which these occurred granular phthisis, in order to distinguish it from tubercular phthisis, in which the small tubercles, for which he invented the name miliary, were soft and tended to break down. He also separated empyema from phthisis, with which it had previously been confounded.

Bayle's grey granulations were subsequently proved to be really tubercles which had undergone fibroid change.

These views were still further developed by Laennec, who claimed a specific nature for phthisis and denied its inflammatory origin. In his opinion all phthisis was tubercular, the tubercles existing either as isolated nodules or as an infiltration, but both alike commencing as a gray and transparent substance, which subsequently became yellow and softened, producing a material like pus, which was discharged from the bronchi, and so led to the formation of cavities.

Laennec regarded caseation as, in all cases, evidence of tuberculosis, and inasmuch as the pneumonia associated with tubercle generally caseated, he considered this to be also of tubercular nature.

This simple view of phthisis was disputed in almost every particular. From Laennec's time up to quite recent date the questions debated were ever the same, viz.—

1. Whether the gray and yellow tubercles were the same in nature, and that too whether they occurred in the nodular or in the infiltrated form.
2. Whether caseation were a change peculiar to, and, therefore, pathognomic of, tubercle.
3. Whether tubercle stood in any definite relation to inflammation, and, if so, what that relation was.

Laennec and his school may be regarded as closing the period of naked-eye observation. It was followed by that of histological investigation, in which careful search was made for structures which should be pathognomic of tubercle, *i.e.*, which should determine what was, and what was not, tubercle.

These Lebert thought he had discovered in his tubercle corpuscles, as Langhans did later in the giant-cell.

Virchow gave a fresh turn to the discussion when he introduced a purely anatomical definition of tubercle by restricting the term to the small miliary granulation which he described as a lymphatic new-growth (lymphoma) prone to caseation. He distinguished this from tubercular infiltration on the one hand, and from caseous pneumonia on the other. Caseation he held to be a general pathological change met with in many morbid products, and not peculiar to tubercle. At the same time he adopted Buhl's views that caseous material was infective and capable of exciting tubercle, and he thus explained the eruption of secondary tubercles round old caseous lesions in the lung.

Virchow's views carried all the weight of his great authority, and largely prevailed, especially throughout Germany, for nearly a generation, but they were never generally adopted in this country nor in France.

Niemeyer, with an exaggeration which was not unnatural, pushed these views to the extreme when he stated that the greatest evil which could befall a consumptive was to become tubercular.

About the time that Virchow was writing most upon the subject, Villemin was opening out an entirely new line of investigation by his inoculation experiments.

Buhl had shown in 1857 that an outbreak of tubercle in the lung was in most cases (90 per cent. at least) associated with pre-existing caseous masses somewhere in the body.

Villemin, influenced by these facts, and by the resemblance which he conceived tubercle to present to syphilis and glanders, was led to the conclusion that tubercle was infective, and put his theory to the test of experiment. He inoculated animals with caseous material, and succeeded in thus transmitting tuberculosis from animal to animal and from man to animal, and that with caseous material derived from any source, *e.g.*, from the lung, lymphatic glands, or elsewhere. Villemin thus proved to his own satisfaction that tuberculosis was a specific infectious disease, and that caseous material, being the vehicle by which the infection was conveyed, was tubercular in nature.

His experiments were frequently repeated by subsequent investigators under varied conditions and with all kinds of animals, with results so generally successful as to lead to the conclusion which Toussaint expressed, that tuberculosis was one of the most communicable of all diseases.

Other observers, however, were led by their experiments to conclude that though caseous substance was the most certain means of exciting tuberculosis, it was not the only one. On the other hand it was shown that these other substances frequently produced no lesion at all, that if they did excite an apparent tuberculosis, this tuberculosis was not communicable, and that by previous disinfection these substances could be rendered inert.

It was also demonstrated that caseous substance was capable of producing tuberculosis, not by inoculation only, but also by inhalation and feeding.

Thus the case for the infectious nature of tubercle became so strong, that in 1879 Cohnheim prophesied the early discovery of the specific virus.

An active search for the tubercle germ was being carried on, notably by Klebs, Aufrecht, and Baumgarten, when Koch in 1882 announced its discovery.

By ingenious methods of staining, Koch succeeded in discovering the bacillus, proceeded to cultivate it outside the body, and proved its specific nature by inoculation, inhalation and feeding.

Up to the time of Koch's discovery, in spite of all the labour expended, the question had hardly advanced much beyond the stage at which Bayle had left it, for there was no criterion by which the tubercular nature of any lesion could be determined.

The specific nature of phthisis having been conclusively established, attention is now being directed to the questions of prophylaxis and immunity, *i.e.*, to the means by which infection may be prevented or the body protected against it, fields of research so rich in promise as to open

out a vision of the time when phthisis may have become as rare a disease as leprosy, once so prevalent, now is.

For some years the problem of tuberculosis appeared solved, but quite recently the question has once more become complicated by the discovery that there are other acid-resistant bacilli, morphologically identical with the tubercle bacillus, which cannot be distinguished from it except by cultivation and inoculation.

Lastly, Koch himself has denied the identity of bovine and human tuberculosis, so that the whole question of the relation of human tuberculosis to that of animals, and especially of cattle, will have to be investigated afresh.

PATHOLOGY.

Phthisis is a specific disease caused by the tubercle bacillus. It is the commonest form of tuberculosis of the lungs, though not the only form, but acute miliary or general tuberculosis has a clinical course and features of its own, so that clinically it is rightly distinguished from what is ordinarily understood as phthisis. Acute miliary tuberculosis might be called tuberculosis peracuta, and phthisis in its various forms described as acute, subacute, or chronic tuberculosis.

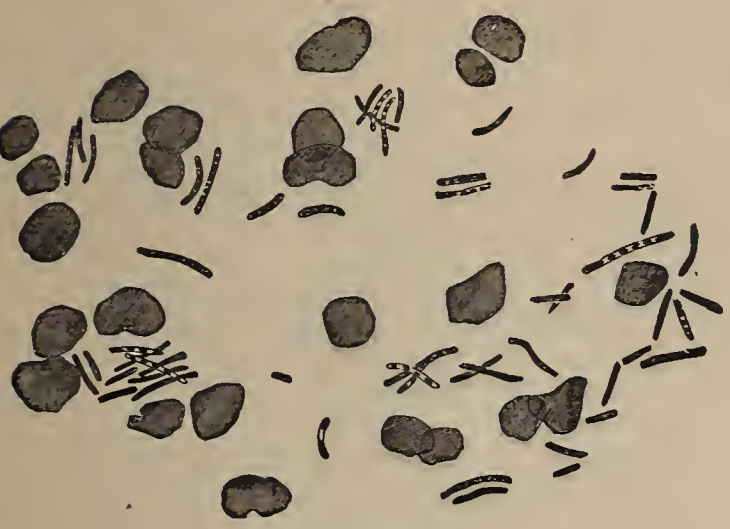


Fig. 103.

Tubercle bacilli from human sputum. (Klein, *Micro-org.*)

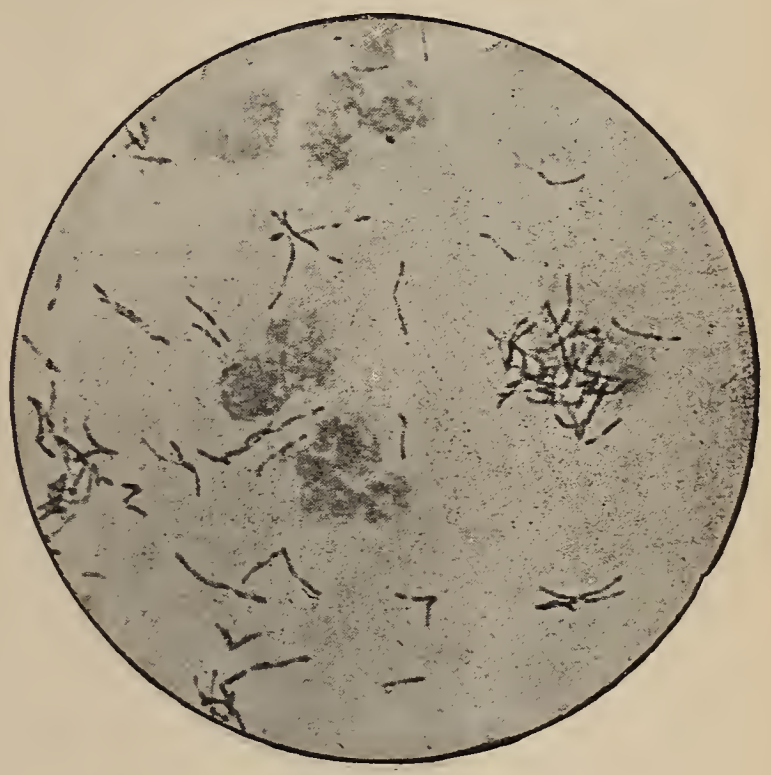


Fig. 104.

Similar specimen. (Klein, *Micro-org.*) Numerous long tubercle bacilli.

The bacillus, when it establishes itself in the tissues, causes in them certain morbid changes, the early ones allied closely to new-growth on the one hand and to inflammation on the other, the later ones being of the nature of degenerations, viz., caseation and fibrosis. These changes are combined with each other in such varying proportions in different cases that the morbid appearances become very complicated, and the relation between them very difficult to determine. It was not until Koch made his great discovery that it became possible to place the various morbid changes in their proper relation to each other, and to study the natural history of the disease.

The bacillus has now been isolated and cultivated outside the body, so that most of the facts of its life are known. Introduced into the body, it has been shown to produce tubercle, and that in both the nodular and infiltrated form, and to result in one of two changes; the one destructive, viz., caseation, a degeneration almost peculiar to itself; the other conservative, viz., fibrosis, the ordinary process by which nature attempts to cure destructive lesions of any kind.

CHANGES PRODUCED BY THE TUBERCLE BACILLUS IN GENERAL.

The tubercle bacilli are slender rods, from 1·5 to 3·5 μ long, *i.e.*, from one quarter to one half the diameter of a red blood cell. They are often slightly curved, and when long may present a beaded appearance. Their recognition depends upon peculiarities of staining. For this purpose the aniline dyes fuchsin or gentian-violet are employed in a watery solution containing alkali, carbolic acid or aniline. The bacilli stained in this way retain the dye even when washed in diluted sulphuric, nitric, or hydrochloric acids, and alcohol, while the colour is discharged from other bacilli excepting those of leprosy. If the preparation so decolorised be now stained by some other colouring matter, the tubercle bacilli are shown in brilliant contrast. The stained bacilli often show in their interior several bright unstained spots which were regarded as spores, but it is now held that the tubercle bacillus does not form spores.

Fig. 105. — Scraping from bovine-tubercle. Two giant-cells, and two small cells containing tubercle bacilli. (Klein, *Micro-org.*)

The staining solutions most used are the following:—

1. *Fuchsin* (Nielsen).	Fuchsin,	1 part.
	5 per cent. watery solution of carbolic acid,	100 parts.
	Alcohol,	10 ,,
2. *Acid* for washing.	Dilute watery solution of sulphuric acid, 25 per cent.	
3. Counter stain.	Methylene blue,	2 ,,
	Alcohol,	15 ,,
	Water,	85 ,,

Fig. 106.—Colonies of tubercle bacilli on the slanting surface of solidified blood-serum (natural size). (From specimen in Dr Klein's collection.)

Method of staining.—The fuchsin solution is warmed till steam rises. The preparation previously dried is placed in it—sputum film for five minutes, section of tissue for fifteen

minutes—and then placed in the acid solution till the colour is discharged (a minute or two). After well washing in distilled water, it is counterstained with methylene blue.

The sputum film when dried may be mounted in Canada balsam. The section must be dehydrated rapidly with alcohol, cleared with xylol, and mounted in xylol balsam.

The tubercle bacilli may be easily cultivated upon solidified blood serum, upon blood serum and gelatine, and upon glycerine agar. If a sterilised tube containing such cultivating media be inoculated from tubercular tissue, and kept at a constant temperature of about blood-heat, the bacilli will grow, and in from seven to fourteen days a dull, white, scale-like growth will appear at the seat of inoculation and slowly spread over the surface, always, however, remaining superficial. From such a cultivation other tubes may be inoculated, and thus many successive generations of tubercle bacilli be obtained.

Growth takes place best at the temperature of blood-heat, and is only possible within a narrow range of temperature, *i.e.*, between 28 and 42 C. (82° to 108° F.). Above and below these temperatures the bacilli will not grow at all. The bacillus is aerobic, *i.e.*, requires free access of air.

When animals are inoculated from these cultivations, tuberculosis is produced, with greater ease in some animals than others, but with success in nearly all. The places chosen for inoculation have been the subcutaneous tissue, the peritoneum, and the anterior chamber of the eye. Tuberculosis has been also produced by injection into the veins, and by the inhalation of a spray containing bacilli.

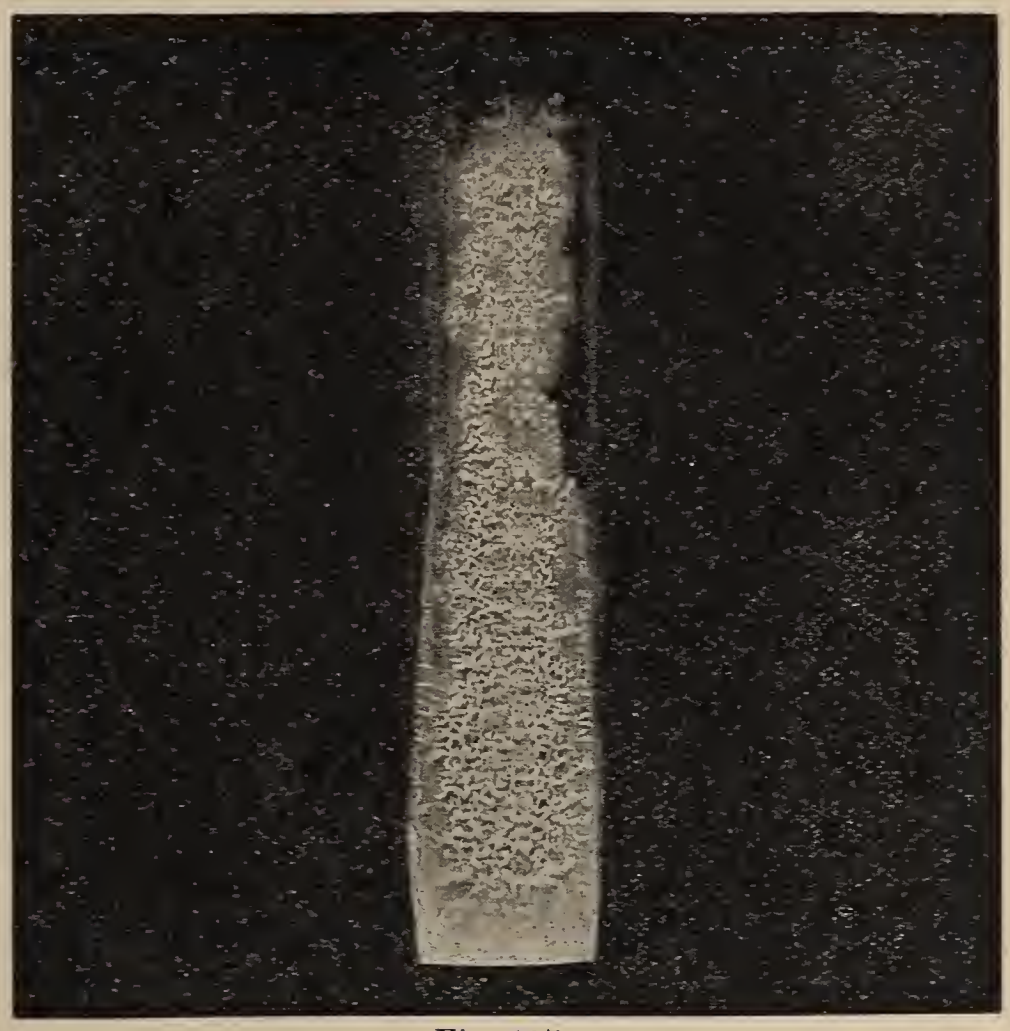


Fig. 107.

Tubercle growth in pure culture on the slanting surface of glycerine agar, showing the characteristic wrinkled, membranous expansion of the growth (natural size). (From a preparation in Dr Klein's collection.)

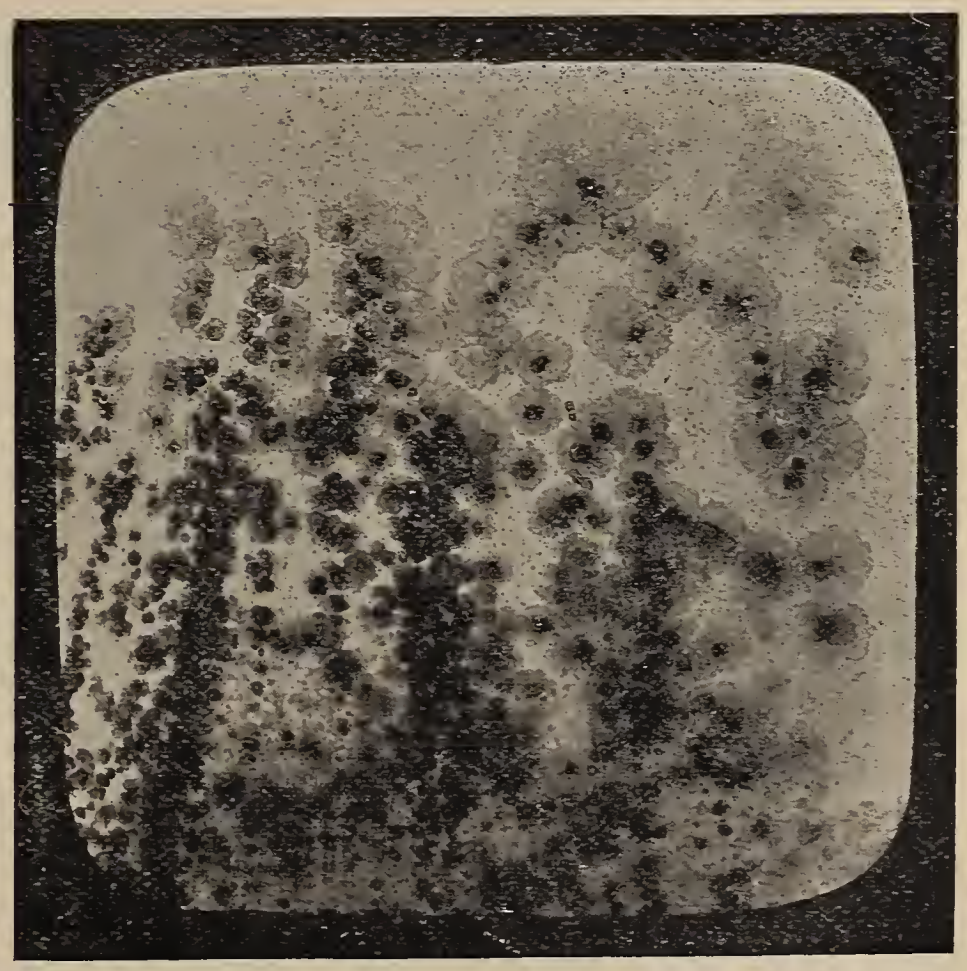


Fig. 108.

The same colonies more highly magnified (× 9).

Tuberculosis being thus capable of production at will, it has become possible to study with accuracy the successive changes which the bacilli excite in the tissues.

The first effect of the development of the bacilli in a tissue is the growth of the fixed cells of the connective tissue, as well as those in the walls of the blood vessels and lymphatics. These proliferate and produce the large so-called epithelioid cells. The bacilli lie for the most part between these cells, but also within them. As the cells multiply they push aside the connective tissue of the part, and it is this which gives the appearance of a reticulum which was formerly regarded as characteristic of tubercle.

Fig. 109.

Two giant-cells and two small cells containing tubercle bacilli. (Klein's *Micro-org.*)

The growing cells contain two or more nuclei, and some of them many, and thus are formed the large protoplasmic multi-nucleated masses termed giant-cells, in which many bacilli are often found.

These foci of epithelial growth are often well defined and sharply marked off from the tissue in which they are formed, but soon inflammatory changes take place round them, and they become surrounded by a zone of small cells, which may become so abundant as to cover up, and even almost conceal, the epithelial cells in the centre. This constitutes the so-called lymphoid tubercle. In some tubercles the small-celled growth is very active and early, and few epithelial cells are to be found in them, but it is possible that in such cases they may develop later.

The tubercle in this stage is a small, gray, transparent cellular mass not larger than a millet seed, and is known as the *gray miliary tubercle.*

As soon as the tubercle has reached a certain size, degenerative changes begin in the centre. First the small cells disintegrate, their nuclei shrivel and break up (tubercle corpuscles of Lebert). Then the large cells disintegrate too; they become pale and homogeneous, lose their nuclei, and

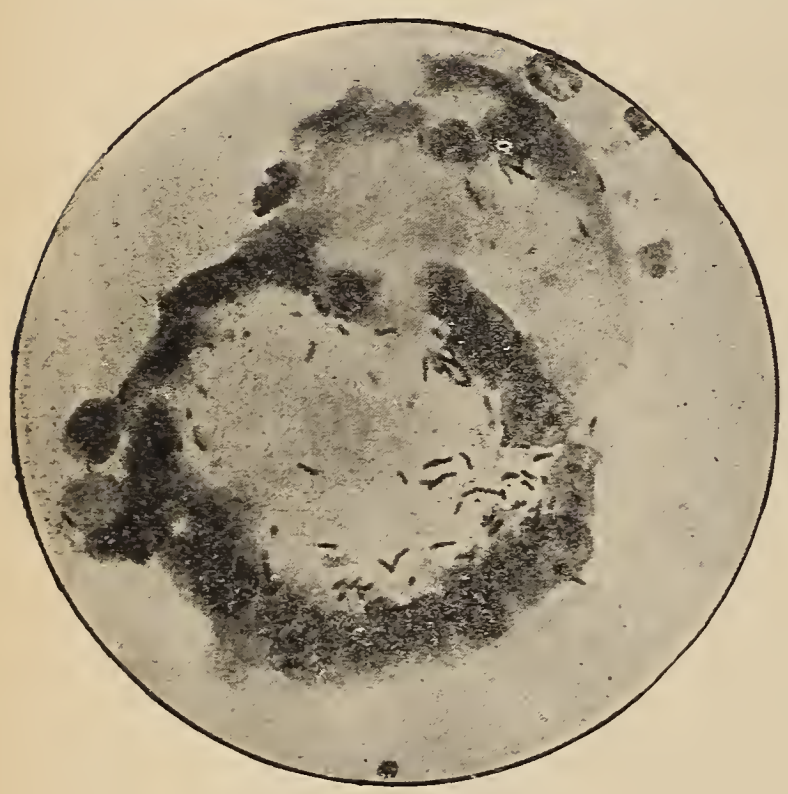


Fig. 110.

Giant-cell (× 1000). The ring-like arrangement of the tubercle bacilli and the nuclei around them is well shown. (Klein's *Micro-org.*)

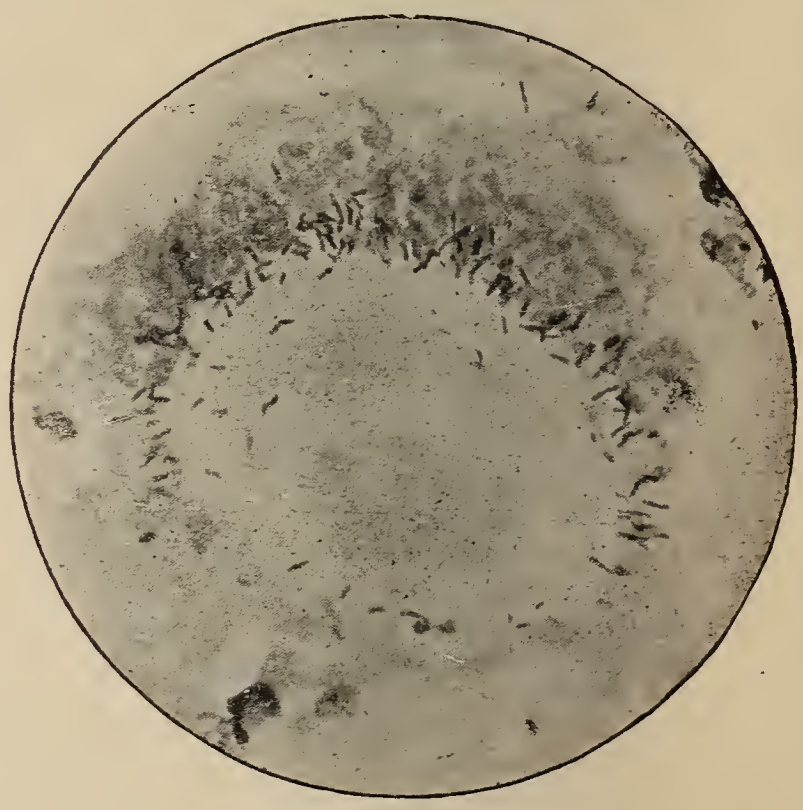


Fig. 111.

A similar specimen (× 1000). (Klein's *Micro-org.*

are converted into hyaline masses. In the giant cells similar partial necrosis sets in, the nuclei being found in the still living parts at the periphery or the centre, as the case may be, and the bacilli at the margin of the necrosing part. In this way the tubercle becomes converted in great part into a uniform hyaline or granular mass, and the necrosed parts cease to take the

ordinary tissue-stains. The tubercle thus loses its gray, translucent appearance, and becomes yellowish white and opaque. It is now described as the *yellow tubercle*, and the degeneration is known as *caseation*, from its resemblance in colour and consistency to cheese.

At this stage the process may stop, the tubercle cease to grow, and the caseous mass become surrounded by small-celled infiltration, from which connective tissue may develop, and form a fibrous capsule. This is the *Encapsulated Tubercle*.

The caseous substance may sometimes be absorbed entirely, and in the end nothing be left but a patch of fibrous tissue. More commonly it is not removed, but becomes infiltrated by lime salts, and calcified (the *calcified* or *cretaceous tubercle*).

Usually the caseous mass softens and breaks down. If this occur upon a free surface, as in the bronchi or intestines, an ulcer is formed, if in a solid organ a cyst containing a puriform detritus. If the cyst be near the surface and burst, its contents will be discharged, and their place taken by air ; thus a cavity is produced such as is common in the lungs.

The essential changes are the same wherever tubercles develop, though they may be somewhat modified by the particular organ in which they are seated.

Tubercle is essentially a local disease, and often spreads for a long time only slowly by direct extension, but if the bacilli gain access to the blood vessels or lymphatics, it may be widely disseminated, or become general.

The lymphatics are always involved early, and the small, recent tubercles which often surround an older caseous mass are of lymphatic origin. The lymphatic glands corresponding with the tubercular part also before long become tubercular. The lymph glands act to the tubercle bacillus, as they do to other pathogenic matters, as filters, and though becoming themselves tubercular, tend thus to keep the affection localised. The glands nearest to the tubercular mass are, of course, most affected ; in other words, the lymphatic affection varies inversely with the distance from the source of infection. If the glands, however, be not competent to strain off all bacilli, some may pass on with the lymph stream to the thoracic duct, and so gaining access to the blood stream, be carried over the whole body, and thus produce an outbreak of general tuberculosis.

The lymphatic glands are, however, sometimes greatly affected without any obvious tubercle in the parts from which they draw their lymph supply. If there be reason to believe that the bacilli have been introduced through these parts, it is clear either that they must have reached the glands without producing any lesion in the tissues, or that the lesion they did produce has completely disappeared.

The most important fact about tuberculosis is this, that so long as there is no dissemination by the lymphatics or blood vessels, its growth or extension is slow, and there is the tendency for it to set up the conservative fibroid change, which may lead to its so-called cure. In this condition the process may remain for a long time stationary, but even a cured tubercle is a source of danger, for the bacilli are not dead, but only quiescent, and thus old tuberculosis may recrudesce and a fresh outbreak occur years after the primary disease has been thought to be cured.

CHANGES PRODUCED BY THE TUBERCLE BACILLUS IN THE LUNG.

The changes produced by tubercle in the lung are the same in kind, though the gross lesions are more complicated and difficult to unravel. For this there are many reasons—

1. The complex structure of the lungs.
2. The inflammatory lesions with which pulmonary tubercle is accompanied.
3. The rapid and extensive excavation which follows.
4. The extension of the process by dissemination through the air-tubes, as well as by the blood stream and lymphatics ; and
5. The secondary infections which take place in the tubercular parts.

The gray and yellow tubercules are as familiar in the lung as elsewhere, but they do not constitute the only or even the most striking part of phthisis. The prominent lesions are the large caseous masses, the patches of pneumonic inflammation, the irregular cavities, and the fibroid induration. These are combined in varying proportions in different cases, and it is the relative predominance of one or other of them that determines the name by which the form

of phthisis is described; thus it may be called acute, pneumonic or inflammatory phthisis, where the caseating pneumonia predominates, or chronic fibroid phthisis where the fibroid induration is most marked.

The gray and yellow tubercles show the same structure in phthisis as in other organs, and do not present greater differences than are met with in experimental tuberculosis.

The gray tubercle shows the same epithelial proliferation and zone of small cells, with giant cells and tubercle bacilli scattered in varying numbers throughout, and there is the same tendency to caseous and fibroid changes.

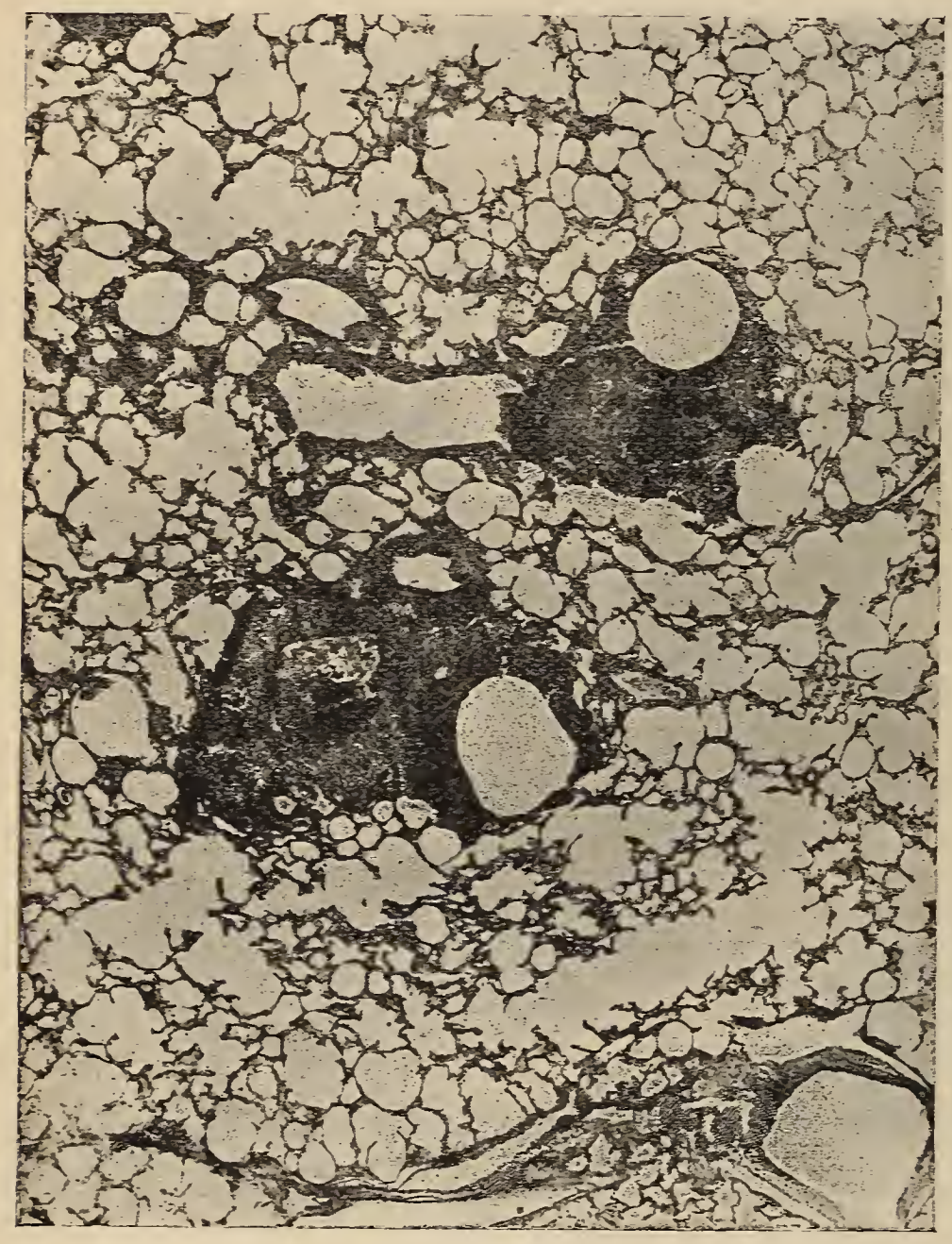


Fig. 112.

Two irregular masses of tubercle. In the centre of the upper is a dark patch, which is a large giant-cell. The masses are formed of interstitial growth and caseous pneumonia. Section taken from the lower lobe of a phthisical lung, the upper lobe being in an advanced condition of tubercular disease.

According to the age and rate of growth, the epithelial and small-celled factors predominate, or else the fibro-caseous changes, and thus are produced the epithelial, lymphoid, caseating, or fibroid tubercles. The yellow tubercles are larger than the gray, and are often composed of conglomerations of the smaller tubercles which have undergone caseation.

Both yellow and gray, when small, are irregular in shape, being seated in the interstitial tissue of the lung in the irregular spaces between the alveoli or bronchioles. The larger yellow masses are rounder in shape, owing to the neighbouring alveoli being filled with consolidation, which also caseates. In some of these cases the alveoli are simply obliterated, owing to the extension of the interstitial growth by a process of budding, as it were, from the tubercular tissue into them, but more frequently the consolidation is due to inflammatory exudation like that of pneumonia, which, because it caseates like the rest, has been called caseous pneumonia. It is to this caseous pneumonia that the rapid increase in the size of a tubercle is for the most part due.

The inflammation often extends a considerable distance beyond the tubercle that excited it, and varies greatly in character and intensity, even in adjacent

alveoli; so that one group of vesicles will present the lesions of catarrhal pneumonia, the exudation containing many epithelial or catarrhal cells; and another group lesions like those of acute croupous pneumonia with fibrinous exudation, and even many red blood cells. The patch, however, hardly ever presents the firm granular surface seen on section in croupous pneumonia, and, when completely caseous, its section is smooth and homogeneous like a piece of cheese or soap. In the early stage this pneumonia often presents a peculiar gelatinous appearance, and has been called on that account *gelatinous infiltration* or *pneumonia (the desquamative pneumonia of Buhl)*. Its peculiar character and its close relation to tubercle were long recognised and insisted upon even by those who did not regard caseous pneumonia as tubercular in nature.

Fig. 113.—Section of caseating pneumonia—tubercular broncho-pneumonia—showing the pneumonic infiltration undergoing caseation, in which the interstitial tissue becomes subsequently involved. (From a preparation in Dr F. W. Andrewes' collection.)

Although this pneumonia may to some extent resolve and disappear, still its natural tendency is towards caseation. Caseating broncho-pneumonia forms a very important element in all cases of acute or subacute phthisis, and in some rare, indeed very rare, cases of acute phthisis constitutes the entire change apparent to the naked eye. Caseating pneumonia has now been shown by experiment, especially by inhalation experiments, to be itself tubercular in nature. The broncho-pneumonia is due, like ordinary broncho-pneumonia, to the irritation of foreign substances in the air-tubes, but the caseation to the presence of the tubercle bacillus. Caseation in simple broncho-pneumonia, or in croupous pneumonia, if it ever occur at all, is extremely rare.

Fig. 114.—Portion of preceding specimen, magnified to show tubercle bacilli, in the caseated substance occupying the vesicles. (From a preparation in Dr F. W. Andrewes' collection.)

But the gray and yellow tubercles and caseating pneumonia do not form the only lesions in early phthisis, for the interstitial tissue of the lung is found greatly thickened and infiltrated with new-growth similar to that of a tubercle in composition and undergoing similar changes. This also has been shown to contain the tubercle bacilli and to be tubercular in nature, so as to deserve the name *Tubercular Infiltration*, by which Laennec rightly described it.

The most striking characteristic in tubercle, other than caseation, is the absence of blood vessels, for no new blood vessels are formed, and the existing ones are involved early in the disease. The tubercular process spreads to the walls of arteries and veins alike, the lumen is plugged with clot, and both walls and clot caseate, so that before long the vessel coalesces with the tubercular tissue round and ceases to be distinguishable from it. These changes in the vessels may lead, on the one hand, to hæmorrhage, and on the other, by providing an easy access for the bacilli to the blood, to the wide dissemination of tubercle through the body.

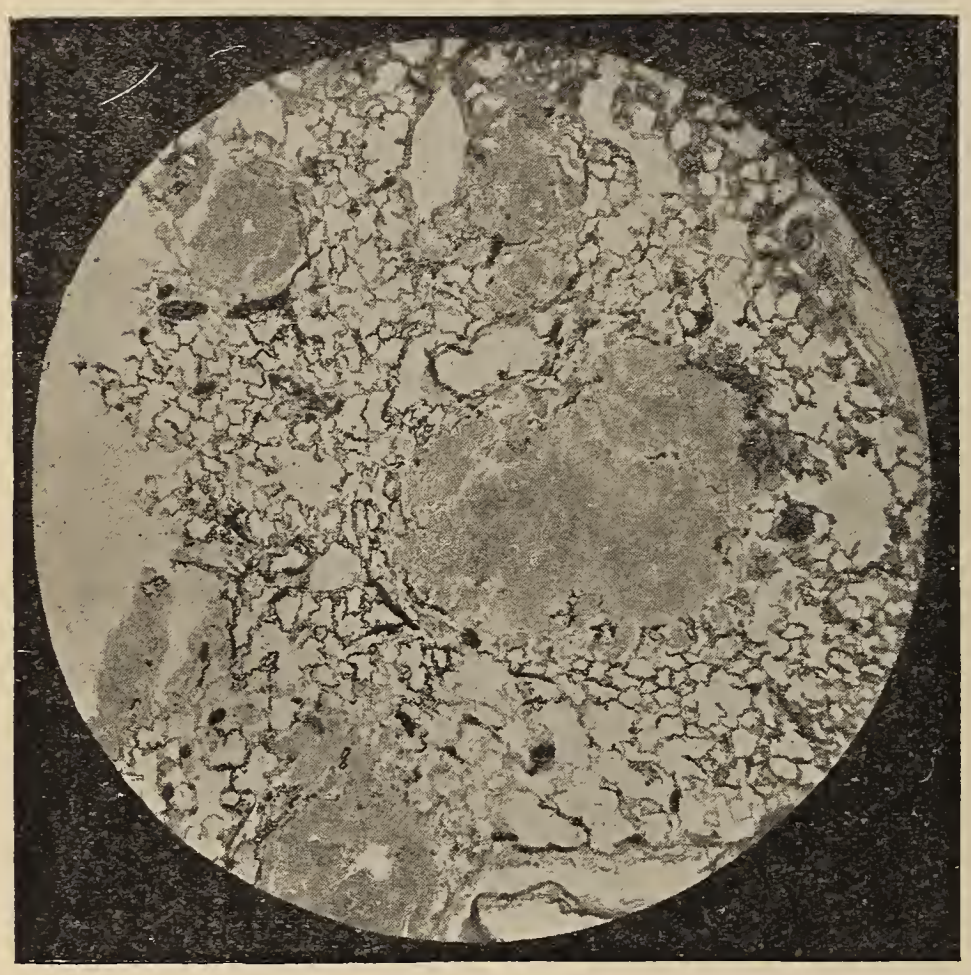


Fig. 115.

Tubercle from an injected specimen, to show the disappearance of blood vessels. (From specimen in Dr Klein's collection.)

The lesions of early phthisis are thus comparatively simple.

The gray or yellow tubercles, the interstitial infiltration, and the caseous pneumonia are all alike the results of the irritation of the tissue by the tubercle bacillus, *i.e.*, they are all tubercular in nature, and they all undergo the characteristic degeneration. The differences between the different forms of acute phthisis depend upon the relative preponderance of the one or the other tubercular lesion.

When a caseous patch softens in the lung it must almost, from the nature of things, communicate soon with the air-tubes, and as the contents are removed by expectoration, air takes their place, and thus a cavity is formed, the walls of which are lined with the disintegrating caseous tissue. It is especially in the walls of such a cavity where the caseous substance is exposed to the air that the bacilli are found, and sometimes in such numbers as to form masses which may, when stained, even be visible to the naked eye. They occur here in greater luxuriance and abundance than is ever the case within the lung tissue.

Cicatrization.—So far the changes described have all been entirely of a destructive nature, but accompanying them is another process of a conservative character which tends to limit the disease, viz., fibrosis. Though not an essential part of tuberculosis, this is the natural process by which cure is attempted, and, when it occurs, achieved. The small-celled infiltration which surrounds the

early tubercle, if it be not itself involved in the tubercular process and caseate, leads to the production of fibrous tissue.

This may occur even in the gray form, and it was these firm gray fibrous tubercles which were first described as miliary granulations, and afterwards known as the *gray granulations of Bayle.* In the same way many of the firm fibrous nodules surrounding the bronchi, and described as Peribronchitis Fibrosa, are nodules of tubercular origin (*tuberculosis indurativa*), and the question may well be raised both upon pathological and clinical grounds, whether some, at any rate, of the cases of diffuse fibroid change in the lung, and which have been called *Cirrhosis of the Lung* or *Fibroid Phthisis*, are not also of tubercular origin, and the result of past and cured tuberculosis. Indeed, Moxon spoke of these cases as phthisis with the age forgotten. The character and duration of the tuberculosis of the lung can be roughly estimated by the amount of fibrous tissue found *post-mortem*, or by the clinical evidence of its presence during life; the acute or recent cases having little, and the chronic or long-standing much.

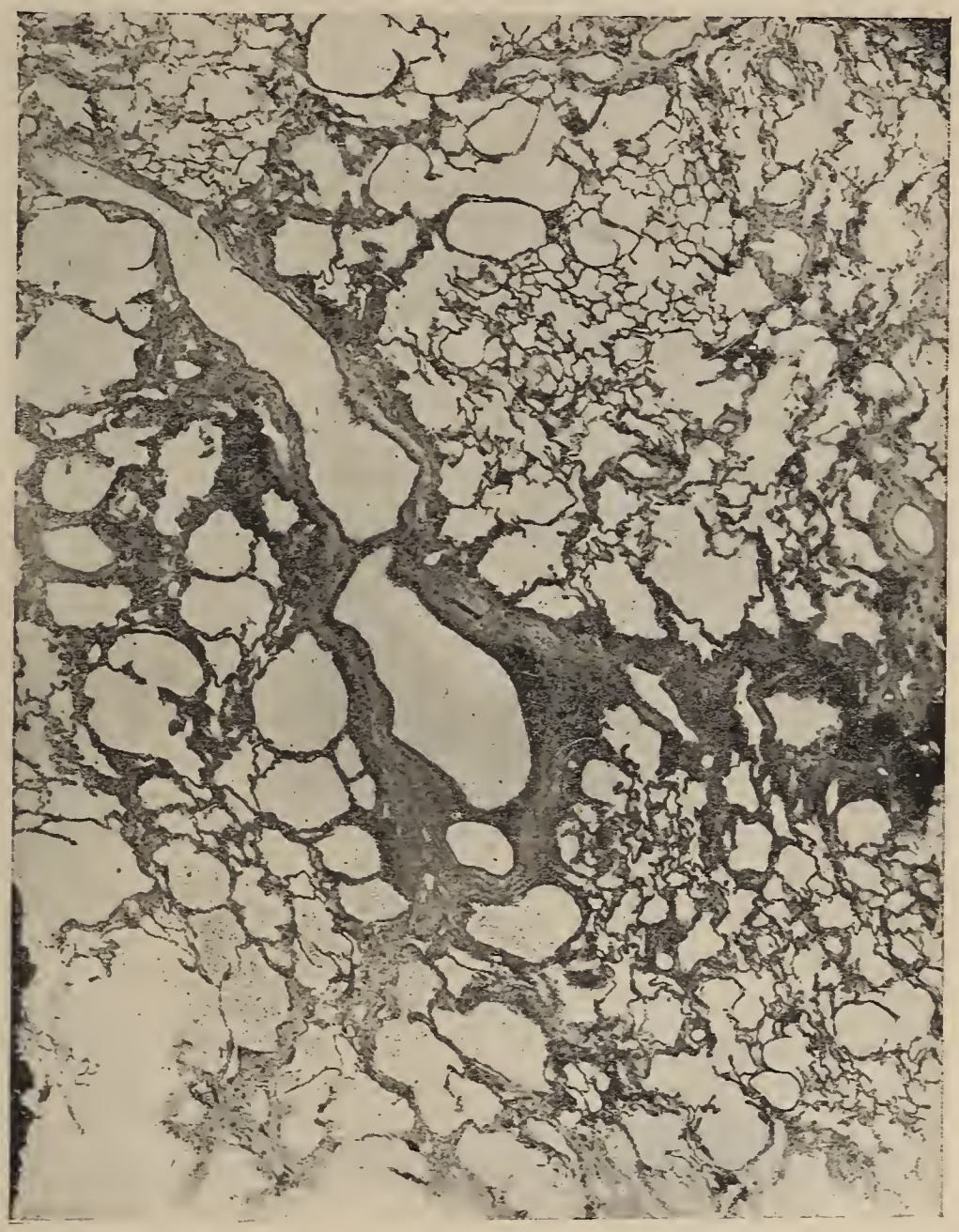


Fig. 116.

Section to show the widespread interstitial fibrosis of the lung in chronic phthisis in parts some distance from the chief seat of lesions.

In chronic cases of phthisis every stage in the development of fibrous tissue can be observed in the same lung, from the immature, imperfect form surrounding recent lesions, to the dense, firm, fully-organised tissue of the older parts. Where the patch of tubercular tissue is small it may be completely encapsuled by fibrous tissue, and the contents become calcareous or cretaceous, or the caseous substance may be absorbed and nothing be left but a puckered cicatrix to mark its site. These two forms constitute the so-called *healed tubercle.* Where a cavity has formed the cicatrization is rarely complete, and though the cavity may be reduced in size, it is doubtful if it can ever be completely obliterated. This could only happen if the cavity were very small, and then it could hardly have been diagnosed during life.

The fibrosis is, as would be expected, most marked where the fibrous tissue is normally in greatest amount, *i.e.*, in the interlobular and interlobar tissue. Where the excavation has progressed far, the outer walls may be formed of the thickened pleura only, while the thickened septa of the lung, or as many as persist, remain as the coarse trabeculæ of chronic cavities. The pleura is, as already stated, very early involved in phthisis, and is a favourite seat of secondary tuberculosis. As the result of this it becomes much thickened and its cavity often obliterated, changes which are really conservative, for they protect the patient to some extent from the risks of pneumothorax and pleuritic effusion.

Cavities.—The most striking feature in phthisis is the excavation that occurs, and on this subject something more must be said. When a caseous mass softens, the degeneration commences in the centre, and thus is formed a kind of cyst containing a creamy fluid, which, though like pus, contains little else but disintegrated caseous substance, with some fat drops, cholesterin crystals, and a few pus cells. This is usually spoken of as a tubercular abscess. The contents may remain for a long time in the fluid state, or, after gradual absorption, become inspissated or cretaceous. More frequently, if near to the surface, the abscess bursts and discharges itself externally. The consequences of the discharge vary according to the organ affected; thus in the intestine or on the skin, a superficial eroded surface is produced, which is described as a tubercular ulcer; in a lymph gland a cavity with a fistulous passage forms, *i.e.*, a tubercular abscess or fistula. In the lung both conditions are met with, tubercular ulcers in the bronchi and tubercular cavities in the lung substance. The smallest cavities in the lung are found in the centre of a caseous mass formed of interstitial tubercular infiltration and caseating broncho-pneumonia. From the nature of things the softened patch will soon communicate with a bronchus and so evacuate itself; as the contents are discharged air takes their place, and so the ordinary air-containing cavity is produced.

The walls of such a cavity consist of three parts—(1) a caseous lining in which all trace of lung structure is lost, the margins of which, towards the cavity, are breaking down; (2) a zone in which the early tubercular changes, both interstitial and alveolar, are seen; and (3) an outside zone of small-celled interstitial infiltration and recent broncho-pneumonia; in fact, the same three layers that are seen in a tubercle.

As the tubercular process extends, the cavity grows in size, and if there be other cavities of similar character in the neighbourhood, they may coalesce, and thus the affected part become honeycombed, as it were, with irregular cavities communicating with each other.

If the process of excavation be less acute or of long standing, the external zone of small-celled infiltration has time to develop into more or less well-formed fibrous tissue. In this stage the cavity will still contain a caseous lining, but it is usually of diminished thickness, as is also the middle zone of tubercular infiltration. If the caseous lining be scraped off, the surface of the tissue below appears red and vascular, and with numerous small hæmorrhagic points.

In very long-standing cavities the fibrous tissue is denser and thicker, and the caseous lining much smaller, or replaced by what resembles a pyogenic membrane, *i.e.*, granulation tissue discharging pus.

Last of all, the discharge of pus may cease and the granulation tissue also be replaced by fibrous tissue, the surface of which towards the cavity is smooth and covered by an epithelial lining. This is the smooth-walled chronic cavity with little secretion, and on that account often called "dry."

In chronic phthisis the fibrous tissue which develops resists the destructive process longest, and forms the coarse trabeculæ which separate adjacent cavities from each other. These trabeculæ have two origins. They are the remains (1) of the thickened interlobar and interlobular septa of the lung, which extend sometimes right across the cavity from the root to the pleura; and (2) of the bronchi and vessels with the thickened tissues round them, and these are most marked towards the root of the lung.

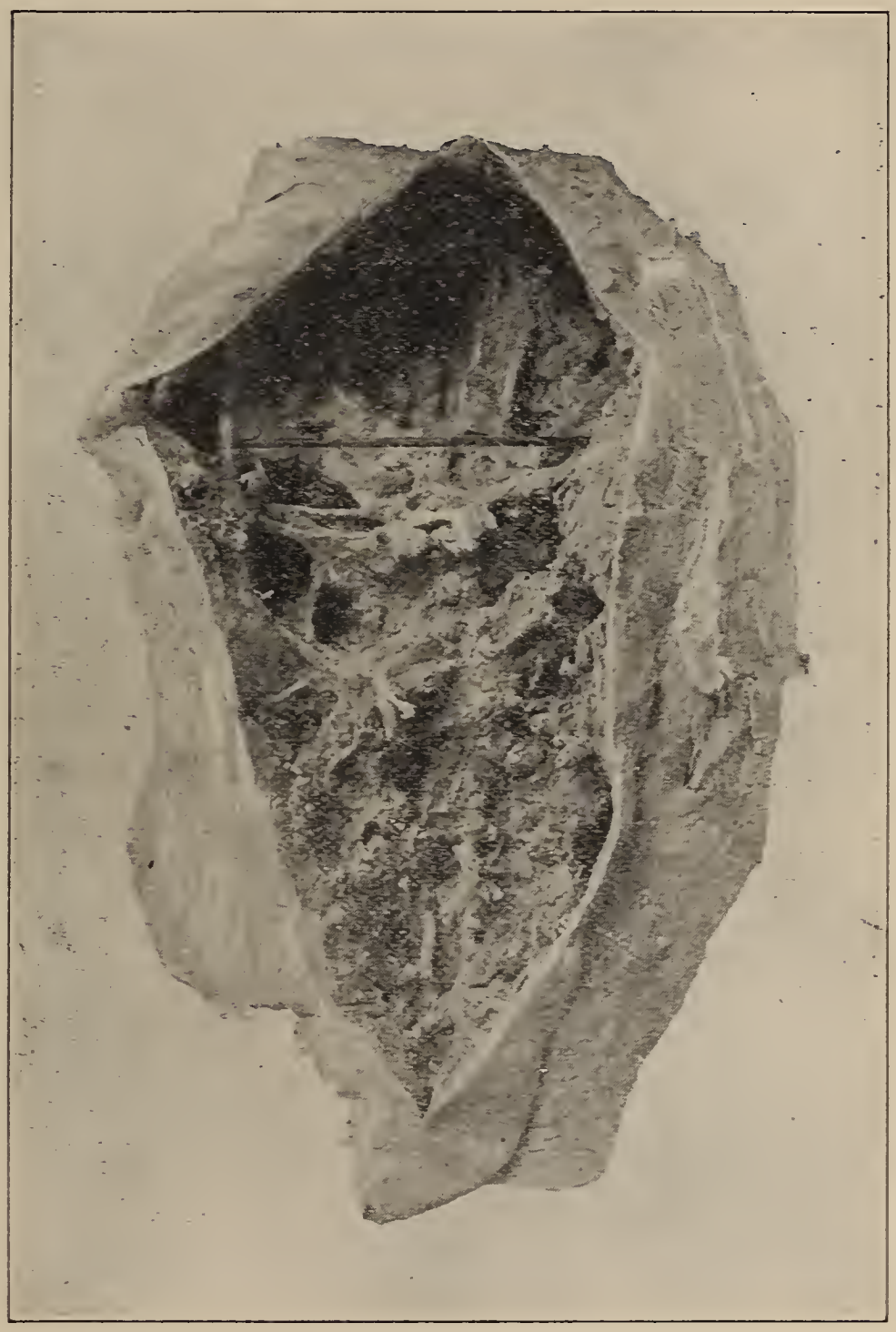


Fig. 117.

Large trabeculated cavity, involving the greater part of one lung —the truncated ends of the bronchial tubes and blood vessels are well shown. (From a preparation in the Museum of St. Bartholomew's Hospital, No. 1723.)

The second kind of trabeculæ are of considerable clinical importance, for they often contain pervious blood vessels which, becoming involved in the tubercular process, may, with or without previous dilatation to form an aneurysm, rupture and cause profuse or even fatal hæmoptysis. Chronic cavities of the kind described may reach enormous size, and occupy a whole lobe or even a whole lung.

So far the increase of the cavity has been regarded as due to the gradual extension of the tubercular process, and in many cases this is slow, or, at any rate, not rapid. It may happen, however, that owing to infection with other germs, suppuration or even gangrenous changes may arise in the walls and around them, and thus lead to a very rapid extension of the cavity. This is rather more common in the chronic than in the other forms of excavation, but it is rare in either.

The chronic smooth-walled cavities are often called *bronchiectatic*. The term was introduced by Laennec, and has been adopted by most writers since, but it is a misnomer, for the smooth lining is not mucous membrane, as it should be if the cavity were a dilated bronchus. Mucous membrane is, it is true, found here

and there in patches, but only where the mouths of small bronchi open into the cavity. Over the rest of the walls it is absent, and it is well established that cavities of other origin may acquire a smooth lining. True bronchiectasis may occur in phthisis, both in the acute and chronic forms, but it is not altogether common, and the chronic cavities described are at the time of death always more than bronchiectasis. The diagnosis of bronchiectatic cavity is a combination of fact and opinion, the fact being that there is a chronic cavity which cannot be disputed, and the opinion being that the cavity has originated in dilatation of a bronchus, an opinion which may be, and often is, erroneous. The bronchiectatic origin of these chronic cavaties, if common, ought to be capable of easy demonstration, for transitional forms ought to be frequently met with. This is, however, not the case.

The bronchi in relation to cavities.—Although it is obvious that when a cavity has reached any size it must have involved many bronchi, still it is remarkable that cavities communicate, as a rule, with but one or two, and even with them the communication is not very free. Many of the affected bronchi are stumpy and truncated, with a mouth which projects like a papilla from the surface, or they run along the walls some little way and open by an orifice which is hardly large enough to admit a small probe. The explanation lies in the fact, that as the bronchi become invaded by the disease, the tubercular infiltration in the early cases, and the fibroid induration in the chronic, surrounds and compresses the tubes and ultimately occludes them. The communication is subsequently made free with one or two of the larger bronchi, by breaking down of the tubercular tissue or by ulceration. The patent bronchi which lead from phthisical cavities are frequently the seat of tubercular ulceration, extending for some little distance along it, and beyond the ulceration recent tubercles are often found in the mucous and sub-mucous coats.

Excavation being the latest stage of the tubercular process, we should expect that the *regional distribution of cavities* would be the same as that of the early lesions of phthisis.

This is shown by Ewart's[1] figures—

At apices,	282 instances.
In dorso-axillary region,	227 ,,
In mammary region,	189 ,,
In sternal region,	61 ,,
At base,	32 ,,

The contents of cavities are, of course, in the early stages the disintegrating caseous substance, mixed with a little pus, perhaps a little blood, and possibly small fragments of lung tissue.

The relative proportion of these constituents to each other varies with the activity of excavation. In the more acute cases there will be a larger amount of caseous substance, more bacilli as a rule, and a greater likelihood of fragments of lung tissue. In the more chronic cases the amount of pus is larger, and it is observed in the sputum that the amount of pus and the number of bacilli often stand in inverse proportion to each other, from which fact the conclusion might be drawn that the cavities have a suppurating rather than a caseating surface, and in this sense the old assertion may be accepted that suppuration is opposed to tuberculosis. Unless there be active suppuration from the surface, the discharge from a cavity is not great, the main part of the sputum being derived from the bronchi, and having the ordinary bronchitic characters. In some

[1] Croonian Lectures, 1882.

chronic cavities the discharge may be almost nil, and the cavity be, as it is termed, "dry."

Many other organisms besides the bacilli may be found in the contents of cavities; for instance, the organisms of suppuration, gangrene, and pneumonia, as well as those of putrefaction.

A remarkable case of this kind is recorded by Bristowe, where a greenish fungus with sporangia was found growing.

Attention has often been drawn to the fact that the contents of cavities in phthisis rarely undergo putrefactive or septic change. The germs which excite these changes must, under most circumstances, be introduced by the air-tubes from without, but for this it has been already shown that the condition of the bronchi is not in many cases very favourable.

Changes in the Cavities and their Contents.—

1. *Increase in size.*—This is usually due to the gradual, and, as a rule, not very rapid, extension of the tubercular mischief; but it may be the result of ulceration or gangrene, and is then rapid and extensive.
2. *Decrease in size.*—This is the consequence of the formation of fibrous tissue in the walls and around. Cavities frequently shrink in this way, but it is hardly possible that any but the very smallest can become completely obliterated.
3. *Putrefaction of the contents.*—This is of rare occurrence, and will be further considered later.
4. *Hæmorrhage.*—This varies in amount. It may not be more than enough to tinge the contents, or in such quantity as to fill the whole cavity with fluid blood and clot. When slight, the bleeding may possibly come from capillaries or small vessels, but copious hæmorrhage is always due to the lesion of a large vessel, either erosion or the rupture of an aneurysm, the latter being the most common in chronic cavities.

SITE.—In all forms of phthisis alike the apices show a remarkable susceptibility. With but very few exceptions, it is at the apex that tubercular mischief commences, and it is there that the oldest and most advanced lesions are found. Even in acute general tuberculosis of the lung the same distribution is observed, though not to the same extent.

The explanations offered are for the most part unsatisfactory.

One is based upon the fact that the movements of the apex are less free than those of other parts; but if this were the reason, we might expect to find a difference between men and women, and no such sexual difference exists.

Another theory connects the defective expansion of the apices with the erect position in man; but Lebert says there is not this special apex-liability in the quadrumana, while it is equally evident in quadrupeds.

The theory that caseation was most frequent at the apex, because the lungs were driest there, may be dismissed as being incapable of demonstration, and in all probability untrue.

The most satisfactory explanation is an anatomical one, which refers it to the different mode of distribution of the bronchi to the upper and lower lobes of the lung. The apical bronchi take a steep direction upwards. As a result of this, the air on passing in and out of the upper lobe is diverted almost at a right angle, and on expiration will meet the air-stream from the rest of the lung in such a way that its passage out is impeded, and if expiration be forcible, as in coughing, may be altogether checked. It is even possible, considering how much greater effect in emptying the air-tubes coughing has upon the lower parts of the lung than upon the upper, that secretion might thus be forced up into the apical bronchi, and then sucked still further in on inspiration.

This explanation, however, is not a complete one, for as a corollary it would follow that infection must usually be the result of aspiration, whereas in acute miliary tuberculosis of the lung, where the infection is probably through the blood vessels rather than the air-tubes, the same predominance of the lesion in the apices is observed.

According to Dr. Kingston Fowler's observations, it is not at the actual summit of the apex that the earlier lesions occur, but at a part 1–1½ inches below it, corresponding in front with the middle of the clavicle. From this part the lesions spread at first chiefly backwards, so that there may be well-marked signs behind when there are few in front.

The second part to be attacked early is the apex of the lower lobe, corresponding with the middle of the interscapular space.

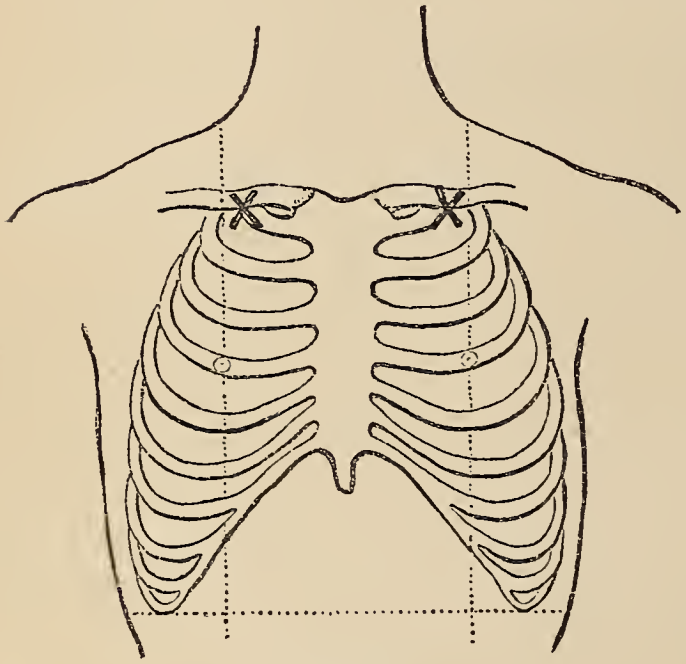


Fig. 118.

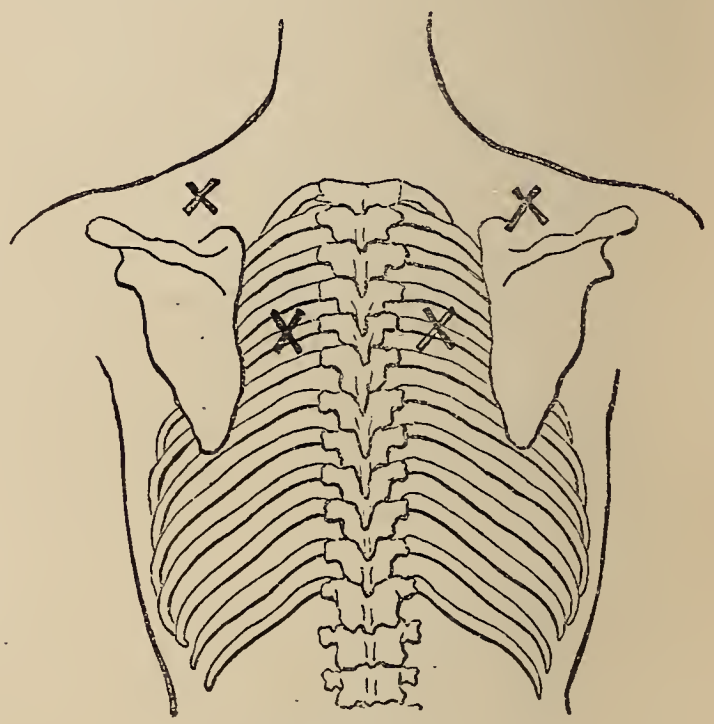


Fig. 119.

Diagrams to show usual seat of the earliest tubercular lesions (after Kingston Fowler).

The lesions spread from above downwards, but the bases are often but little affected, and may escape altogether. Basic phthisis, *i.e.*, phthisis in which the bases alone are affected, or affected first, is very rare.

The lesions spread to the opposite lung in the same order, but Fowler maintains that a favourite early spot for secondary infection is the middle of the interlobar septum, corresponding with a spot in the upper part of the axilla.

Basic phthisis is a term that is often used, yet, strictly speaking, it is a rare condition.

Physical signs at the base are not uncommon in phthisis. They are chiefly due to some pleuritic complication, *i.e.*, chronic pleuritic thickening or effusion, or perhaps a localised empyema; in other instances there may be some non-tubercular affection of the base of independent origin, *e.g.*, chronic pneumonia with its results.

By basic phthisis is implied tubercular disease, which is either limited to the base, or which, at any rate, commenced in the base.

In the majority of cases in which base signs exist alone, or predominate, it is found on *post-mortem* examination that, though the lesions at the base are most recent and most active, there are old antecedent tubercular lesions in the apex which may be regarded as the source of infection. As it is impossible by physical examination to exclude the existence of old arrested

lesions in the apex, a diagnosis of basic phthisis which rests on clinical signs alone, and is not confirmed by *post-mortem* evidence, is necessarily inconclusive.

Primary or pure basic phthisis occurs, but only in an extremely small percentage of cases, possibly not more than 1 per cent., if so much.

Louis gives 2 cases in 123	Kidd gives 1 case in 60–80
Walsh ,, 2 ,, 1000	Cotton ,, 2 cases in 412

Kidd[1] gives an interesting case of primary symmetrical basic phthisis which ran its course in nine months.

I have seen 2 cases of the same kind, both bilateral, in which I was unable even by the most careful physical examination to detect any signs in the upper lobes, but as in neither case was a *post-mortem* examination made, it was impossible to prove the absence of old apex lesions.

Sides Affected.—At the time of death both sides are involved. To this rule there are but few exceptions. In the early stage, however, it is very rare to find both apices attacked at the same time, though it cannot be proved by figures that there is any decided preference for the one side over the other. In the published statistics, the distinction is usually not clearly made between the lesions of healed tuberculosis and those of active phthisis, for the lesions of healed tuberculosis are localised, and often unilateral.

Although it is true that both sides are affected sooner or later, still there is often at the time of death a great difference in the extent of the disease upon the two sides, and that is especially the case where there is extensive excavation. In this respect complementary emphysema may be taken as evidence that the hypertrophying lung is at any rate not the seat of active mischief, for, when in the opposite lung disease is active, no hypertrophy occurs.

Mode of Spread.—It is important to consider the way in which tubercular disease, once developed in the lung, is likely to spread.

Tuberculosis is essentially a local affection, and though liable at any time to dissemination, it may for a long time remain local, spreading it may be only slowly by direct extension into the surrounding parts. If it become disseminated, it may spread by the lymphatics, the blood vessels and the air-tubes.

(*a*) *Infection by the lymphatics.*—In the pulmonary tissue surrounding a caseous mass early tubercles are often seen, both gray and yellow. These are the results of lymphatic infection, and are especially common in the subpleural tissue, which is abundantly rich in lymphatics. The lymphatic glands at the root of the lung are involved early, and rarely if ever escape entirely. They may be of considerable size, and are sometimes enlarged out of all proportion to the amount of local mischief in the lung. They may even be affected without any obvious tubercular lesion in the lung at all; from which it may be argued that the bacilli, if they have not gained access to them in other ways, have passed through the lung without producing any lesion there, or else that the lesion they did produce has entirely disappeared. Secondary tuberculosis of lymphatic origin is not widely disseminated, but more or less restricted to the parts in the neighbourhood of the primary lesion, or between this lesion and the root of the lung.

(*b*) *Infection through the blood vessels.*—This will lead to a much wider dissemination either through the body at large, *i.e.*, to general tuberculosis, or, if confined to the lung, throughout both lungs. The blood vessels in the neighbourhood of tubercular mischief are involved very early, and their lumen usually obliterated, either by the direct extension of the tubercular process or by thrombosis. At any rate, they soon undergo the same degeneration, and

[1] *Lancet*, 1886, p. 615.

are lost in the caseous mass. All the vessels alike are involved, both pulmonary and bronchial, arteries and veins. The coats of even the large vessels or main

Fig. 120.—Section of recent acute tuberculosis of the lung from a case of phthisis. Tubercles of all sizes are seen disseminated throughout the lung. Many lie close under the pleura. The larger masses are seen from their texture to be largely formed of broncho-pneumonic consolidation. Extensive infiltration is seen surrounding, and extending along, the air-tubes and blood-vessels.

trunks may become the seat of tubercle, as in instances already mentioned. In these and other ways, it is easy to see how caseous substance or the bacilli them-

selves may gain access to the blood. They will then be carried, like small emboli, as they really are, with the blood stream, and scattered over the body. Wherever they stick the bacilli may grow, and give rise to fresh tubercles. Now it is common, as the result of tubercular infection, to find recent tubercles widely scattered through both lungs, but by no means so common to meet with general tuberculosis. For general dissemination throughout the body, the infective emboli must find access to the left side of the heart so as to be carried by the arteries, but this can only occur when the infection has found entrance into the pulmonary veins. If the infective material has entered by the pulmonary artery, or by the bronchial artery or veins, it is to the lungs that it will be first carried, and there filtered off. If this be so, there will be three chances to one that when blood infection through the blood occurs, it will lead to pulmonary rather than to general tuberculosis. The matter is not really as simple as this, for it is evident that the size of the bacilli is such that they could easily pass through the capillaries of the lung, and thus be disseminated over the body at large; still it is clear that this does not always or necessarily occur.

(*c*) *Infection by the air passages.*—It is this which is responsible for the most rapid spread of the disease in the lungs. When a cavity has formed, and is discharging its contents into the air-tubes, there is the ever-present danger that some of it may not be expectorated, but be drawn by aspiration into the tubes of healthy parts, and there set up a broncho-pneumonia, in which the tubercular changes will occur. It is this form, caseating broncho-pneumonia, that artificial tuberculosis takes in animals, as the result of the inhalation of tubercular spray. The same occurs, no doubt, in man, and accounts for the rapid progress of some cases of phthisis. But if tubercle bacilli be really as infectious as is sometimes supposed, and inhalation-infection as easy as is sometimes assumed, it is extraordinary, not that aspiration-pneumonia should sometimes occur, but that it does not always occur, and cause all cases of phthisis to run a rapid course, instead of the chronic course that most take.

Secondary tuberculosis, however excited in the lungs, runs through the same course as the primary lesion if life be prolonged, and may itself become the source of fresh infection.

The widespread tuberculosis in the lungs is, however, generally miliary and still gray when the body is examined. This is to be explained, no doubt, by the fact that the sudden outbreak of fresh tubercle has caused such an exacerbation of symptoms, that the patient dies before the tubercles have had time to undergo the usual changes.

How often the lungs are the seat of primary tuberculosis, or how often they become affected secondarily as the result of infection from some caseous focus elsewhere in the body, are questions that must be reserved for subsequent consideration.

ASSOCIATED PATHOLOGY.—The pathological changes with which phthisis is liable to be associated fall into two groups, viz., those which are met with in the respiratory organs and those which occur in other parts of the body.

A. Changes in the Respiratory Organs.

1. In the Lung Tissue.—*Congestion.*—In acute cases those parts of the lungs which are not directly involved in the tubercular process are in a condition of congestion owing to the collateral hyperæmia consequent upon the destruction of vessels in the tubercular parts and the diminished aerating surface. In chronic cases the collateral circulation adjusts itself and congestion is not marked.

Complementary hypertrophy.—When contraction occurs in the tubercular parts the neighbouring alveoli undergo complementary dilatation or emphysema. It is usually rather a hypertrophy than an emphysema, for the distended parts are capable of increased physiological activity and not of less. Complementary hypertrophy is most marked in cases of chronic phthisis affecting one lung only, and it may be so considerable that the enlarged lung may extend two or three inches beyond its normal situation at the middle of the sternum. The hypertrophied lung is itself either quite free from tubercular mischief, or shows nothing but some very limited and old lesion, but it may become the seat of a fresh outbreak of tubercular mischief after a time.

Suppuration and gangrene.—Reference has been already made, when speaking of cavities, to their extension by suppuration in the walls and round them. This, however, when distinguished from the results of tubercular mischief, is with little doubt the result of independent infection by the organisms of suppuration, and stands in the same position as gangrene. Both are rare developments in phthisis, and will be further discussed under the head of complications.

2. **In the Air-tubes.**—*Bronchitis.*—We should expect the bronchi to become affected both on account of the collateral congestion and because of the irritation produced by the passage of the tubercular sputum over them. General bronchitis, however, is by no means common in phthisis, unless it occur as an independent complication. As a necessary part of the disease it is met with only in acute and widely disseminated tuberculosis, and is then the result of the collateral congestion. Local bronchitis is almost invariable and may be the earliest manifest lesion before the tubercle, which has caused it, gives other evidence of its presence. It is due partly to the congestion, partly to the irritating discharges, but also in part to the local development of tubercles in the coats of the bronchi themselves.

Bronchiectasis.—Bronchiectasis is not uncommon both in the acute and chronic forms, being in the one case the result of nutritive changes in the walls whereby the resistance is diminished, and in the other the mechanical effect of the contraction of the fibrous tissue around.

Bronchial atresia.—Obliteration of the air-tubes is, as already stated, common in the walls of cavities, the occlusion being due in early cases to the tubercular infiltration and in the latter to the contraction of the fibrous tissue surrounding the tube.

Secondary Tuberculosis of the Air-tubes.—This may be brought about on the one hand by the direct extension of tuberculosis from the neighbourhood, or by infection from the tubercular sputum.

Spreading by contiguity is most frequently seen in the small bronchi leading from cavities, but it may be met with in any air-tube, large or small, with which a tubercular mass is in contact; besides this the peribronchial tissue is itself a favourite seat of eruption for secondary tubercles, from which the process may extend to the bronchial coats and lead to perforation and the formation of ulcers.

The lesions of the air-tubes produced by infection from the surface are superficial erosions, and follicular or more diffuse ulcerations. The erosions are small, punched-out, ulcer-like patches resembling the aphthous ulcers of the mouth, surrounded by an area of redness or congestion, and discharging a little pus in which tubercle bacilli are often found.

The tubercles are for the most part of small size and not easy to be distinguished, except by microscopical examination, from swollen and inflamed follicles. They are sometimes larger, and may form yellow masses prominent above the surface.

The seats of tubercular ulceration are in order of frequency (1) the small bronchi leading from cavities; (2) the larynx; (3) the trachea; (4) the larger bronchi.

An analysis of nearly 3000 cases collected from various sources shows the larynx to have been affected in about 33 per cent., and among these the trachea was also involved in about 25 per cent., but it is rare to find the trachea alone involved. This occurred in only 1·5 per cent.

The larynx.—It is in the larynx that tubercular ulceration assumes its chief importance. It is found at the time of death, as already stated, in fully one-third of all cases of phthisis, and some writers place its frequency even as high as 50 per cent. In many of the cases the lesions are slight, and would be easily missed, unless carefully looked for. The frequency of laryngeal mischief must not be judged by symptoms, for even extensive ulceration may produce few symptoms if it does not involve the vocal cords, while hoarseness or loss of voice may be due to simple laryngitis or muscular failure, without any ulceration at all.

When ulceration exists the vocal cords are most frequently the seat of mischief, either alone (33 per cent.) or with other parts of the larynx (39 per cent. more). Of the other parts of the larynx, the epiglottis and subglottic region are but rarely affected alone; the latter, according to Lebert, in 4·8 per cent. of all cases of ulceration.

The most striking feature in laryngeal tuberculosis is the great swelling, or, rather, dense infiltration which often develops. This usually stands in direct relation with the ulceration, but not necessarily, and it may be considerable without any ulceration at all. The infiltration is composed of inflammatory tissue in which tubercles are abundant. Actual stenosis of the larynx is, however, in spite of the swelling, rare. Stenosis such as to necessitate tracheotomy only occurs in about 1 case in 1000.

If the tubercular disease reach the cartilages, suppurative perichondritis may be excited, and necrosis of the cartilage with all its disastrous consequences result.

The infiltration may sometimes be localised and assume tumour-like characters. These conditions are known as *tubercular polyps* or *tubercular vegetations*, and though met with in almost every part, their favourite seat is in the anterior part of the larynx between and above the true vocal cords.

Though at the time of death tubercular disease of the larynx is almost always found associated with phthisis, and in the great majority of cases the clinical history proves conclusively that the laryngeal mischief has followed that in the lung, still there is now no fair ground for doubt that tuberculosis of the larynx may be of primary origin, and persist, even for a long time, without pulmonary complications.

The whole subject has been already fully discussed in a previous chapter, to which reference may be made.

The epiglottis is rarely involved except in connection with tubercular ulceration of neighbouring parts. The ulceration usually spreads from the larynx, and as it first affects the base or sides, it may be invisible during life, owing to the considerable swelling and immobility of the parts with which it is associated.

Lebert states that in 13·5 per cent. the epiglottis is affected alone, but this percentage is much above the usual experience.

3. Affections of the Bronchial Glands.—The bronchial glands are always involved more or less in phthisis, especially those which surround the main bronchi at the root of the lung, and those which lie in the mediastinum at the

bifurcation of the trachea. In chronic phthisis they are fibrous and deeply pigmented, and in acute phthisis they are greatly enlarged, soft and caseous. In children the enlargement is often out of all proportion to the amount of tubercular mischief in the lung, and in some cases there is no evidence of tubercle to be found in the lung at all.

When greatly enlarged, the glands may press upon the trachea, bronchi, œsophagus, or even on the vessels, and may then give rise to dyspnœa, cough, stridor, or dysphagia.

From the glands the tubercular process may extend to any of the organs mentioned, and when softening occurs the contents may be discharged into them. When this discharge takes place into the air-tubes, the risk of tubercular broncho-pneumonia and the outbreak of rapid phthisis is very great, and if a vessel be involved, acute tuberculosis, either pulmonary or general, may be the result.

These and other complications have already been discussed.

4. In the Pleura.—*Tubercle.*—The lymphatic tissue beneath the pleura is, as already described, a favourite and early seat for the development of secondary tubercles, and pleurisy is the natural result. The pleurisy may be acute or chronic, but in either case it leads generally to obliteration of the cavity, and is not, as a rule, attended with effusion. It usually extends far beyond the main focus of mischief in the lung, and though, like this, most common at the apex, it may be general and cause complete obliteration of the pleural cavity. It is often attended with considerable fibroid thickening, which may be so firm that the lungs cannot be removed without the use of the knife. It is the contraction of this tissue that is the chief cause of the flattening and defective movements in the side affected. In this fibrous tissue vessels may develop, and anastomosis be thus formed between the vessels of the lung and those of the walls of the chest. This manifests itself in dilatation of the subcutaneous vessels over the affected part.

It may happen that this new tissue may itself become tubercular, and thus the process extend across the pleura to the walls of the chest, and end in producing an external fistula.[1]

Chronic abscess of the chest wall is not altogether rare in phthisis. It may be of quite independent origin, and when opened may take long to heal. Such an abscess might not be easy to diagnose from the fistula described, or from a localised discharging empyema.

The pleurisy being secondary to the tubercles of the lung, and both lungs being, as a rule, involved sooner or later, it follows that the pleurisy will often be bilateral, and being, as stated, not restricted to the original seat of mischief, it may be widespread. In some cases of disseminated tuberculosis general bilateral pleurisy is the earliest sign of disease.

These facts explain the gravity attached, and rightly, to bilateral pleurisy, for it is generally secondary to some bilateral cause, and that cause is frequently tuberculosis.

When the pleura is the seat of primary tuberculosis in man, caseous masses of considerable size may be produced, as in cattle.

Pleuritic effusions are not uncommon, and may be either serous or purulent.

Serous effusions are more important as clinical complications, for they are rarely the cause of death. *Post-mortem* evidence is somewhat misleading in this respect, for the serous effusion found after death has often developed during the last few days of life, and is then rather of the nature of dropsy than of inflammation. Such dropsical effusions occur in about 3 per cent. of deaths from all causes (Lebert), and allowance must be made for this in statistics.

[1] *Berl. klin. Woch.*, 1892, Nos. 20, 21.

Two points deserve notice in respect of the serous effusions of tubercular pleurisy; the first is that they are frequently blood-stained or hæmorrhagic, and the second that the fluid often coagulates spontaneously when removed from the body. Hæmorrhagic effusions are rare in simple pleurisy, but are, as a rule, associated with some grave cause, viz., either tubercular or malignant disease.

Empyema, though a much rarer complication of phthisis than serous effusion (in the proportion of 1 to at least 4 or 5), is much more serious, for it is often the cause of death.

The frequency of effusions in phthisis is stated as follows by different authors:—10 per cent. by Louis and Dittrich; and by Lebert as 10 to 12 per cent. for chronic, and as 20 per cent. for subacute phthisis; of hæmorrhagic effusions, as 5·6 by Frerichs and 2·3 per cent. by Dittrich; of purulent effusions, as 1 per cent. by Dittrich, and by Lebert as 2 per cent. for the chronic and 5 per cent. for the subacute; but many of these latter were localised.

Pneumothorax.—In at least 90 per cent. of all cases of pneumothorax the cause is to be found in phthisis, and it is the actual cause of death in phthisis in about 5 or 6 per cent. of all cases.

Lebert's figures give 5 per cent. for all deaths from phthisis. In 326 *post-mortems* of phthisis, I met with 19 instances of pneumothorax, and this yields a percentage of 6·1. Kidd's figures yield 11 per cent.

Pleurisy and pneumothorax are of chief importance as clinical complications, and will be more fully considered hereafter.

B. Changes in other parts of the Body.

1. **The Digestive Tract.**—The *stomach* and *intestines* are very frequently found in a state of catarrh, which may be acute and widespread.

In one case, that of a woman, besides intense gastric catarrh, the mucous membrane of the whole small intestine, from the duodenum nearly to the cæcum, was in a condition of pulpy gelatinous infiltration, and below this, from the last few feet of the ileum to the end of the sigmoid flexure, was the seat of almost continuous tubercular ulceration.

Tubercular ulceration of the intestines is found in from 75 to 80 per cent. of cases of phthisis at the time of death. It is in most cases secondary to the lesions in the lung, and is doubtless caused by infection from the sputum which has been swallowed.

As an isolated affection, tuberculosis of the intestine is very rare, and met with almost without exception in children.

A few cases of this kind, 7 only, are recorded by Barthez and Rilliet, and 2 by Lebert, and I have seen one such case myself.

Such figures are sufficient proof of its rarity.

The *favourite seat* of tubercular ulceration of the bowel is the lower part of the ileum and the neighbourhood of the ileocæcal valves, and in these parts it is usually found, if anywhere; but it may be widespread and extend throughout the whole intestine, large and small. It becomes less frequent in the jejunum, where it is not common, while in the duodenum and stomach it is extremely rare.

Frerichs gives the following figures:—Ileum affected alone in 35 per cent., and together with the colon in 45 per cent.; the colon alone in 3 per cent., and the rectum alone in 7 per cent. Lebert gives 42 per cent. for the small intestine alone, and 7 per cent. for the large intestine alone.

Though tubercles may develop in any part of the intestine, the favourite seats are the Peyer's patches and the solitary follicles, where they commence as small prominences, which soon caseate, soften, and break down. In this way ulcers are formed with thickened infiltrated walls, in the base and neighbourhood of which fresh tubercles form.

	Soltau Fenwick and Dodswell,[1] 500 cases.		Kingston Fowler.[2]
Small intestine alone,			8·3
Large intestine alone,			16·5
Both large and small together,			75·1
Duodenum,	3·4		2·1
Jejunum,	28·0		21·5
Ileum,	85 per cent., alone in 9·6		58
Colon—Vermiform appendix,			41·7
Cæcum,			58
Ascending,	51·4		
Transverse,	30·6		41·7
Descending,	21·0		
Sigmoid flexure,	13·5		
Rectum,	14·1		13·6

The early ulcers are small and round, the later ones irregular in size and shape. In the small intestine they tend to spread transversely round the bowel, following the course of the lymphatics and blood vessels. In the cæcum and at the ileocæcal valve they are quite irregular. In the colon they are often round, and constitute one of the forms of follicular ulceration here, but in rare cases the mucous membrane may be destroyed almost as extensively as in dysentery. In the large intestine, after the cæcum, a common seat of the ulceration is the rectum. The appearance of a tubercular ulcer of the bowel is, as a rule, characteristic. The irregular margins, the thickened and infiltrated walls and base in which caseous tubercles may be recognised, make mistake impossible; if any doubt existed, examination of the peritoneal surface would set it at rest, for in the subperitoneal tissue, round about the seat of ulceration, caseous tubercles would rarely fail to be seen.

When the sub-peritoneal surface is reached, inflammation may be excited in the peritoneum, just as it is in the pleura, with the result generally that the neighbouring coils of intestine become adherent to one another. The chronic peritonitis, thus excited, remains as a rule localised, so that chronic general peritonitis is quite a rare condition in the bodies of phthisical patients, and peritoneal effusions are equally rare.

In my 326 autopsies, diffuse chronic peritonitis was met with 3 times only, *i.e.*, in less than 1 per cent.

In 1 other case there was a collection of half a pint of pus in the peritoneum. Dropsical, as distinguished from inflammatory effusions, are of course not uncommon.

If the adhesions be not firmly formed, or, having been formed, give way, perforation will take place, but it is not a frequent complication.

[1] *Lancet*, July 16, 1892.

[2] Fowler and Godlee, *Lungs*, p. 358.

It occurred in 2 cases out of my 326.

In Fenwick and Dodswell's [1] series it occurred in about 1·2 per cent. (*i.e.*, in 26 out of 2000 cases), and thus agrees with the statement of Dittrich and Louis.

Localised peritonitis is most frequent in the neighbourhood of the cæcum, and not a few of the cases of typhlitis and perityphlitis prove to depend upon tubercular disease of the bowel, or of the vermiform appendix. In the rectum, tubercular disease may lead to abscess or fistula. Tubercular ulcers of the intestine rarely if ever heal completely, though they may cicatrise in parts. This partial cicatrization, together with the peritoneal adhesions, may in some cases produce stenosis or stricture of the bowel, but this is a much rarer complication than might be supposed, and though it may cause some trouble during life, yet it rarely leads to complete obstruction and death.

Tubercular ulceration, as already stated, becomes less and less frequent as the distance from the ileocæcal valve increases. It is not common in the jejunum at all, and is rarely met with in the upper parts.

In 2 out of my series of 326, it was found within 4 feet and 2 feet respectively of the duodenum, and once in the duodenum itself.[2] In this case there were two small ulcers seated 2 inches from the pylorus. This case deserves record on account of the extent of the ulceration, and as being a case in which perforation caused death.

The patient, a woman of 46, had an old cavity in the left upper lobe of long standing. Elsewhere, throughout both lungs, there were numerous recent caseous masses. The larynx was extensively ulcerated as well as the epiglottis. The peritoneum contained about a pint of dark coffee-coloured fluid, similar to what was found in the intestines. The fluid had escaped into the peritoneum through a perforation in the floor of an ulcer, about 4 feet from the ileocæcal valve. Almost the whole intestinal tract was ulcerated. In the duodenum, 2 inches from the pylorus, were two small tubercular ulcers. Ulcers became more and more frequent throughout the jejunum, and from the upper part of the ileum to the rectum there was hardly a piece of healthy mucous membrane left. In the ileocæcal region and in the rectum the mucous surface was occupied by one continuous ulcer. There was no general tuberculosis, and the last point of interest is that the patient suffered from mitral stenosis.

Small quantities of blood are sometimes passed with the motions, especially when there is ulceration in the lower part of the large intestine. The amount is rarely large, but it may be copious or even fatal. Of this rare result Kidd [3] records two instances.

Duodenum, Stomach, and Œsophagus.—Tubercular ulceration of the duodenum, stomach, and œsophagus is very rare.

Of the duodenum, Wilson Fox [4] gives only 10 cases, 2 of them from his own experience, and to these may be added the instance just described.

Of the stomach, Andral and Wilson Fox have met with it once or twice, but Louis never saw it at all. Cless found 1 instance out of 500 cases, but Frerichs [5] met with it 6 times in 250 cases of phthisis.

The most remarkable case of the kind is recorded by Litten,[6] in which a tubercular ulcer occurred in the stomach and in no other part.

Blumer has recently recorded a case of his own, and collected 30 cases from literature.[7]

Of the œsophagus, Frerichs records 1 case; Weichselbaum another, and refers to a third.

Most of the instances of tubercular ulceration of the œsophagus are due to the spreading of ulceration from the neighbourhood, as from caseous glands, for example. It is to this group that the 6 cases recorded by Louis belong, who, by implication, almost denies its occurrence in any other way.

In the same way, ulcers in the stomach may arise by extension from without, *i.e.*, from tubercular masses in the subperitoneal coats, of which instances are given by Lebert and Litten.[8]

[1] *Lancet*, July 16, 1892.
[2] *Cf.* Dittrich, who records 2 cases; quoted Wilson Fox, p. 608.
[3] Clifford Allbutt, *Syst. of Med.*, vol. v. 517.
[4] *Cf.* Wilson Fox, *loc. cit.*
[5] Frerichs, *Lehre d. Tuberculose.*
[6] *Virch. Arch.*, 1876, vol. lxvii.
[7] *Albany Med. Annals*, Mar. 1898.
[8] *Volkmann's Samml.*, No. 119.

Bucco-pharyngeal tuberculosis, *i.e.*, tuberculosis of the lips, mouth, cheeks, tongue, palate, fauces, and pharynx.

Tubercular ulceration of these parts, though a rare complication of phthisis, is hardly ever met with except in that disease.[1] It is the result either of direct extension to these parts from tubercular ulcers in the neighbourhood, or of independent infection from the sputum.

Ulceration due to direct extension is found, as would be expected, in the pharynx and fauces, or at the root of the tongue; and except when produced in this way, tubercular ulceration of these parts is the rarest of conditions.

In independent infection, the ulceration is usually preceded by some simple lesion which becomes infected. It occurs in those parts where such lesions are most frequent, *e.g.*, at the commissure of the lips, at the tip and sides of the tongue, or on the cheek.

If the affected part be excised, scraped, or otherwise actively treated, and partly cured, in most cases fresh infection occurs, and the disease returns.

The ulcers present the ordinary appearances, modified by the fact that they are constantly bathed by saliva, and kept clean by the movements of the mouth. They show a grayish yellow surface, with irregular eroded base and margins, and more or less of infiltration around. In the immediate neighbourhood of the ulcer are usually to be seen small superficial sharply-cut aphthous-like spots which are fresh degenerating tubercles, and which ultimately coalesce with the ulcer.

These lesions are usually met with in the late or terminal stages of phthisis, and are of the gravest omen, for life rarely continues long after their appearance.

In acute tuberculosis, whether consequent on phthisis or not, miliary tubercles have been found in the bucco-pharynx, but they are of pathological interest only.[2]

The diagnosis has to be made from syphilis and malignant disease, especially from the latter, but, as a rule, the suspicion of the tubercular character of the lesion is confirmed by the history of the case and by the physical signs of phthisis.

In some instances the diagnosis is rendered certain by the demonstration of tubercle bacilli in the material obtained by scraping the surface of the ulcer.

The lips.—The ulcer usually starts at the commissure and originates in a crack. It may then spread to the cheek near.

I have seen two or three cases of this kind. The most marked instance occurred in a man of 40, the subject of very rapidly advancing phthisis. The ulcer rapidly reached the size of a sixpenny piece, and was attended with considerable infiltration round, so much so that the question of epithelioma was raised, and on account of the doubt as to its nature, as well as because of the pain and distress produced, its removal was decided on. This was successfully accomplished, and the wound healed, except in one small part on the inside of the mouth, which became reinfected. In a few weeks the condition was as bad as before, and the pain and distress as great as ever, so that the operation proved to be of little service.

The cheeks are rarely the seat of ulceration, unless by extension from the lips or fauces. If the central parts of the cheeks are affected, it is generally to be referred to some erosion started by decayed teeth.

The gums are perhaps on the whole the rarest seat of tubercular ulceration in the mouth. Stomatitis and gingivitis are not uncommon in bad cases of phthisis, and the spongy, ulcerated condition of the gums due to this affection may not at first be easy to distinguish from tubercular ulceration, but the course of the case, and the usually rapid cure under simple remedies, is sufficient for the diagnosis.

[1] *Cf.* Spillmann, *Tuberc. d. Tube Digestif.*, Paris, 1878, with references to literature.
[2] *Volkmann's Samml.*, 1897, 119, p. 15.

Palate.—I have seen two instances of this, both in women.

In one it was due to extension from the fauces to the soft palate, and in the other the ulcer was of independent formation, and situated upon the centre of the hard palate.

A case[1] is recorded by Mr. Battle, in the *Lancet* for Nov. 30, 1889; and another by Fairlie Clark, *Tr. Pathol. Soc.*, xxvii., in a man 18 years of age, in which the soft palate had been entirely destroyed.

Fauces.—This is almost invariably the result of extension from the neighbourhood, especially from the larynx and epiglottis.

Tongue.—It is in the tongue that most instances of tubercular ulceration of the mouth are met with. The common seats are the tip, sides or under surface; the dorsum, and especially the posterior part of it, are hardly ever involved except by direct extension.

An excellent instance of the latter form is recorded by Bowlby,[2] in which nearly the whole of the fauces and the parts connected with it were involved.

At the tip and sides the ulcers commence as small aphthous-like patches, but they soon involve the deeper tissues and then lead to fissures which are the cause of great pain. The tissues around are firm and infiltrated, but not to the extent which is usual in epithelioma, and in this slighter amount of infiltration, as in the usual absence of enlargement of the glands beneath the jaw, are found points of difference which distinguish tubercular from epitheliomatous ulceration.

Like malignant disease of the tongue, tubercular ulceration occurs twice as frequently in men as in women, but in both at an earlier age. As a rule, whatever doubt may have existed at first is set at rest by the history and course of the case, and by its association with phthisis.

A very good account is given by Butlin in his work on the Tongue.

A series of cases are recorded in the *Path. Soc. Trans.*, vol. xxxiv., xxxv., xxxvi., xxxix., and xliii.

The first case in this country was described by Paget in 1858.

Butlin recommends excision, but the risk of fresh infection is very great in phthisis, and as cases in which tubercular disease of the mouth develops usually run a rapid course to death, little is to be gained, I think, by operation, except, it may be, partial relief of pain for the time. Although there may be exceptions, I doubt if, as a rule, as much is not lost by operating under such unfavourable conditions as is gained by any temporary relief given, but each case must be settled on its own merits.

In cases of primary tuberculosis of the tongue, there can be no doubt as to the wisdom of immediate excision, but such cases are extremely rare.

2. Affections of the Liver.—The changes in the liver associated with phthisis are three, viz., fatty infiltration, amyloid disease, and tubercle.

Fatty infiltration is by far the most frequent, and is met with in nearly one-third of all phthisical cases at the time of death.

Murchison gives 30 instances out of 52 cases; Wilson Fox, 26 out of 91; and Lebert in 33 per cent.

It has been commonly attributed to the administration of cod-liver oil and other fatty substances in the treatment of phthisis, but it was described by Louis before this treatment was in vogue. It has been also connected with the emaciation, but it does not occur with the same frequency in other wasting diseases. It might be referred to the diminished combustion of carbohydrates

[1] Gee, *St. Barth. Hosp. Rep.*, vols. vii., xxi.

[2] *Path. Soc. Trans.*, xxxv. 160.

in the lung, but, if so, there should hardly be so much general emaciation. It is, in fact, the contrast between the disappearance of fat in other parts of the body and its excess in the liver which creates the difficulty in finding an adequate explanation.

Amyloid disease.—This is to be connected with the prolonged suppuration in the lung, but being a general disease, it is not the liver alone that is affected, but the spleen, kidneys and intestines as well.

In my series amyloid disease occurred in 20 cases, or 6·2 per cent., and this is the same percentage as that given by Welch.[1] Wilson Fox's is very much higher, viz., 15. As the number of his cases was not large, 91 only, this may have been accidental.

Amyloid disease in phthisis seems to be less frequent than it used to be in phthisis, but there are no figures to prove this.

Tubercle.—When general tuberculosis complicates phthisis, the liver hardly ever escapes. The tubercles are then numerous, but very small, almost microscopic. Tubercles of greater size, as large as a pea or bean, are sometimes but only very rarely found, and in a few instances softening has occurred in them and cavities have been formed.

A very remarkable case of this kind is recorded by Hector Mackenzie,[2] in which the part of the liver affected was riddled with cavities, and the condition resembled closely that which is usually seen in chronic tuberculosis of the kidney. I have also seen[3] a similar case—but there I think the tubercle reached the affected part of the liver by direct extension through the diaphragm.

Cirrhosis.—The association of cirrhosis with phthisis is probably accidental, for although tubercle may lead to interstitial hepatitis in animals, *e.g.*, rodents, nothing of this kind is met with in man except in the rarest instances, *e.g.*, in a case recorded by Orth,[4] where the presence of tubercle in the liver was combined with a growth of dense fibrous tissue as firm as scirrhus.

In my series cirrhosis was only found in 4 cases, *i.e.*, in 1·2 per cent., a percentage probably not above the general average. The liver, it may be noted, was in all these cases much above the normal weight, weighing 70, 82, 86, and 104 ounces respectively.

There may possibly be some connection between phthisis and chronic alcoholism, for some cirrhotic patients die of phthisis, and phthisis is certainly a commoner cause of death in peripheral neuritis than would be expected. At the same time it must be borne in mind that phthisical patients, especially in hospital classes, do not infrequently take to drinking.

3. Affection of the Spleen.—The spleen is a favourite seat of secondary tuberculosis; the tubercles may be small and gray, or caseous and of some size. In acute phthisis the spleen is enlarged and soft, as in septic diseases generally, and when amyloid disease develops the spleen is more frequently affected than any other organ except the liver.

4. Affection of the Heart.—The heart is often small, and no doubt shares in the general atrophy of the body which takes place; but this is not constant, for the heart may be much above its ordinary weight, and that too without any obvious disease in it.

Thus in 20 male and 20 female cases, taken at random, the heart was found to average $10\frac{1}{3}$ ounces in the males and $7\frac{3}{4}$ in the females, *i.e.*, it was a little above the normal weight in the males and a little below in the females; but among the male cases there were no less than 6 instances in which the weight was above 12 ounces, and that without any gross cardiac disease.

[1] *Cf.* Wilson Fox, p. 620.
[2] *Cf.* Morley Fletcher, *Path. Soc. Trans.*, 1899.
[3] *Path. Soc. Trans.*, vol. xli.
[4] *Virch. Arch.*, vol. lxvi.

The cavities of the heart are not infrequently found filled with decolourised *ante-mortem* clots, which may extend from the cavities into the vessels, but in this there is nothing peculiar to phthisis, for it may occur in any case which has been long a-dying. The relation of phthisis to morbus cordis will be considered in another place.

Endocarditis has been described in cases of phthisis, but it is very rare, and when present is, as Biondi[1] has shown, very rarely tubercular, but due to coccal infection.

The pericardium often contains a little serous fluid, which is exuded during the last few days of life and is dropsical in nature.

Pericarditis is rare. Is met with in an acute form sometimes in acute tuberculosis, and is then associated with the presence of tubercles; in phthisis, except as the result of acute tuberculosis and acute pericarditis, it is rare; but chronic pericarditis is rather more common.

It was present in my series in 3 cases, *i.e.*, in 1 per cent., and was due to the spreading of the inflammation from the pleura.

There are a few remarkable cases recorded in which *Pyo-pneumo-pericardium* has been produced by the opening of a phthisical cavity into the pericardium.

5. Affections of the Veins.—Thrombosis of the femoral vein is not uncommon as an accidental complication of the later stages.

6. The General Condition.—The tissues of the body do not seem to be specially prone to decay in phthisis, for patients are, it appears, rather less subject than other chronic invalids to bed-sores, and after death the body resists putrefactive change longer than is the case in many other diseases. These facts were observed by Laennec.

ETIOLOGY.—Now that phthisis has been proved to be a germ disease, the main question in its etiology has been settled. All the other facts in its etiology have now to be considered in relation with the prime cause of the disease, viz., the bacillus. The history of the bacillus within the body, and the morbid changes it leads to, constitute the morbid anatomy of the disease, and have already been discussed. The etiology has to deal with the history of the bacillus outside the body, the ways in which the germ gains access to the body, the means the body has of resisting its attack, and the influence of external conditions, on the one hand upon the offensive power of the germ and on the other upon the defensive power of the body.

Being a germ disease, phthisis is in the nature of things infective, *i.e.*, communicable; that it should also be infectious in the common acceptation of the term is also possible, but does not necessarily follow. Whether it really is infectious or not cannot be assumed, but must be settled by evidence and not by presumption.

History of the Tubercle Bacillus outside the Body.

The tubercle bacillus is of comparatively slow growth, and requires for its development ten to fourteen days when left undisturbed in a suitable soil. It grows best at or about the body temperature, and will not grow at all except within a limited range of temperature, *i.e.*, between 80° and 106° F.

The essential conditions, viz., rest, moisture, suitable temperature and soil, are not likely to be met with in nature except within the body of a warm-blooded animal, so that growth, *i.e.*, multiplication outside the body, is practically

[1] *Ctbl. f. allg. Path.*, 1893, Feb. 23.

impossible. Still, though growth is unlikely, the bacilli are remarkably resistant, and retain their vitality for long periods.

Phthisical sputum can be dried and the bacilli still retain their vitality for forty to fifty days (Koch)[1], or if kept warm and moist, for even as long as nine or ten months (de Toma).[2] They may be heated to 60° C. for twenty minutes (140° F.), or to 70° C. for ten minutes (158° F.), without being completely sterilised.

Exposure for from fifteen to twenty days to maceration in water, to putrefaction, or to repeated alternate freezings and thawings, will not always destroy them.

On the other hand, resistant as the germs are, it is difficult to preserve their vitality beyond the forty to fifty days, unless special precautions are taken, so that this may be regarded as the natural limit of their life. Again, putrefaction and excessive moisture, as well as prolonged drying, tend to their destruction. Direct sunlight and fresh air act on them as they do on anthrax germs and rapidly sterilise them. Koch[3] showed that the tubercle bacilli were killed in a few minutes if in a thin layer, and in a few hours if in a thicker layer, when exposed to direct sunlight, while they perished in ordinary daylight in five to seven days.

The bacilli, therefore, outside the body do not multiply, nor do they under ordinary circumstances retain their vitality for more than two months, and the very process which would most likely lead to their wide dissemination in the form of dust is one of the most active agents in their destruction.

Considering the enormous numbers of bacilli (over twenty millions daily it has been calculated) which are discharged from the lungs even of a single phthisical patient, it is evident that their destruction must be much more active than we sometimes assume or are able to prove.

Sources of Infection.—When we consider the great number of phthisical persons living, and the large amount of bacilli-containing sputum they discharge daily, it is but natural to regard man himself as the chief source of infective material. The sputum, so carelessly expectorated here, there and everywhere, will, when dried, be scattered widely with the dust, and be taken, with the air, into the mouth and lungs, so that it is impossible to avoid the conclusion that this is, at any rate, the ordinary mode of infection in man.

The dust collected from hospital wards, out-patient rooms, dwelling-rooms, and the streets has been proved by experiment to be infective. The bacilli themselves have been demonstrated in the dust and in the deposits on the windows of sick rooms, and in the ventilating shafts of consumption hospitals. In a recent investigation, Coates[4] found in fourteen out of twenty-one houses (*i.e.*, in 66·6 per. cent.) living and virulent tubercle bacilli in the dust of rooms occupied by phthisical patients, the dust being collected not from the floor, but from the walls and ledges, where it had deposited itself naturally from the air in the room.

On the other hand, Cornet[5] showed that in 147 different observations the dust was infective in 40 and non-infective in 60 cases; that the infectivity depended upon whether phthisical persons had been in a position to infect the dust, and that, where this was not the case, the dust was not infective. He concluded that phthisis was only dangerous through the sputum, and not then unless the sputum was dried; that the sputum, being hygroscopic, was very difficult to dry, and yet, to be disseminated with the dust, must be practically desiccated; lastly, that even infective dust often failed to infect, the bacilli being intercepted by the cilia and entangled in the mucus of the healthy air-tubes and so expelled.

Besides the sputum, the discharge from tubercular lesions, *e.g.*, tubercular abscesses, tubercular ulceration of the bowel, etc., are possible, though not likely, sources of infection.

In considering the pathogeny of phthisis in man, *i.e.*, its causation or mode of production, this fact must be clearly borne in mind, that phthisis is very frequently the consequence of tubercle in some other part of the body, *e.g.*, the

[1] *Virch. Jahrb.*, 1886, i. 285.
[2] Cornil and Babes.
[3] *Brit. Med. Jour.*, Aug. 16, 1890. For other micro-organisms, *cf.* Downes, *Trans. Roy. Soc.*, xl., 1886, etc.
[4] Tuberc. Congr., London, 1901.
[5] *Zeitsch. f. Hygiene*, 1889, p. 191.

glands or the bones; in other words, that phthisis is often not the primary lesion, but a secondary lesion. When tubercle exists in any part of the body, the lungs are almost certain to become affected sooner or later if life last long enough, or, as Laennec put it, with tubercle in any part of the body, if any other organ become secondarily involved, it is almost certain to be the lungs. Phthisis, then, is often the result of auto-infection, so that the date of the commencement of phthisis is not necessarily, or, as a matter of fact usually, the date of the introduction of tubercle into the body.

Buhl proved long ago that phthisis was associated with the presence of caseous material in some other part of the body, antecedent to the phthisis; and the teaching of his time might be aphoristically expressed "no phthisis without an antecedent caseous (*i.e.*, tubercular) focus." Buhl's observations are as true now as they ever were. It follows, therefore, that in many cases the seeds of phthisis have been sown long before, and that the patients have become phthisical because they were already tubercular. If this be so, the date of the original infection with tubercle is thrust further and further back, even, it may be, into early childhood, so that, in considering the causation of phthisis, it will be necessary to deal with the original sources of infection in early life.

The liability to tubercular infection no doubt persists throughout the whole of life, so that a patient may become tubercular at any age, even though advanced in years. At the same time the liability decreases as age advances, and it is probably not far from the truth to assert that an adult will probably not become phthisical unless already tubercular.

Thus in little children it is especially the glands that suffer, and of these by preference the bronchial and mesenteric glands; a little later the cervical glands are the common seat of lesion; still later the bones; while it is not until early adult life that phthisis becomes prevalent.

It would almost seem as if the liability of the different parts of the body to tubercular infection varied with the age.

The cases of phthisis thus fall into two etiological groups:—

1. Those in which the pulmonary tuberculosis is primary, *i.e.*, in which the lungs are the chief and the most recent, if not the only, seat of tubercle.
2. Those in which the pulmonary tuberculosis is secondary, *i.e.*, a consequence of pre-existing tubercle elsewhere.

The relative frequency of these two groups cannot be precisely stated, but Buhl's observations show how large the second group is, and it may safely be asserted that it includes the great majority of all cases.

Given tubercle pre-existent in the body, the lung may become affected (1) by direct extension, *e.g.*, from one of the bronchial glands; (2) by the discharge into the air-tubes of material from a caseous gland, which explains some of the most rapid forms of tubercular disease; or (3) by infection carried by the lymph stream or by the blood current.

Tuberculosis in Children.—In children, phthisis, in the ordinary sense of the term, is rare, though they are very liable to tubercular affections of other kinds.

Tubercular meningitis is essentially a disease of early childhood. The acute *broncho-pneumonia* of children is also often tubercular. Both affections are frequently secondary, and found associated with softening caseous glands, of date long antecedent to the acute affection.

The primary seats of tubercular infection in children are the lymphatic glands; the bronchial or mediastinal in the very young, and the cervical in older children.

In many of these cases these glands, of the one or the other set, are the only tubercular lesions found in the body.

Among the children of the poor, caseous bronchial glands are extremely common. Voelcker, in a series of autopsies at the Children's Hospital, Great Ormond Street, found these glands caseous in no less than 33 per cent. Out of 13 cases of tubercular meningitis under my own observation, caseous bronchial glands were the only other tubercular lesion in 8 and caseous mesenteric glands in 2.

The general frequency of tubercular infection of these glands in children is shown by the following figures of different observers :—

	Woodhead.	Rilliet.	Simmonds.	Colman.	Carr.	Average.
	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Bronchial, . . .	75·6	79	73	79	80	76
Mediastinal, . .	78·7	46	46	66	65	60

The relative frequency of primary tuberculosis in the bronchial and mesenteric glands is differently stated by different writers, but on the whole the bronchial glands are more frequently affected than the mesenteric.

When the bronchial and mesenteric glands are the seat of primary tuberculosis, *i.e.*, when there is an accompanying lesion of the air-tubes or of the intestines, it follows that the bacilli must have passed through the mucous membrane without affecting it, reached the lymphatics, and thence been carried to the glands. Fortunately, in many cases, the changes excited there remain local, become quiescent, and the lesions produced become in time encapsulated, and thus shut off from the rest of the body.

Where the bronchial glands are the primary seat of lesion, the infection must have been introduced by the air, and, in the case of the mesenteric glands, with the food. But the distinction is not so sharp as it might seem, for much of the infected dust would be deposited, like all other dust, in the mouth or pharynx, and swallowed.

If it reached the lung, it would be entangled in the mucus, swept back again by the cilia, and finally expelled by coughing. Those bacilli, also, which are swallowed are acted on by gastric intestinal juices, which inhibit their growth and activity, and ultimately destroy them. The body has thus considerable power of protecting itself against the inroad of bacilli, and, so long as the number which gain access to the body in a given time is small, they are expelled without harm. If a few gain an entrance, they are interned in the lymphatic glands. It is only where the numbers introduced are immense that local lesions are produced. This is well seen in cases of phthisis where the sputum is swallowed ; in such cases secondary intestinal tuberculosis is common, and the condition is analogous to the experimental production of intestinal tuberculosis in animals.

If the identity of tuberculosis in animals and man be accepted, other sources of infection obviously exist in food, especially in the *milk* and *flesh* of cattle, and thus the purity of the milk and meat supplies becomes of the greatest importance.

In *cold-blooded animals* tubercle seems to be an extremely rare affection, but it has been described as occurring in reptiles in confinement.

In *warm-blooded animals* it is widely prevalent, and probably all this class are susceptible. Many wild animals, although rarely attacked while living in the normal state, fall ready victims in confinement. This is notably the case with monkeys, rabbits, and guinea-pigs, the last-named being so susceptible that they are usually selected for experimental purposes.

In Cattle.—It is among domesticated animals, and especially among cattle, that tuberculosis is most prevalent.

The returns from different countries show great variations in its prevalence, but agree in showing it to be very common in every one.

In Berlin, where systematic examination has been carried on for many years, the percentage of tubercular cattle for all cattle slaughtered is 3·4. In Great Britain and Ireland it is stated to be about 4. Bollinger calculates that not less than 5, and probably as much as 7 or even 8 per cent., is the average rate for milch cattle, and that in all probability, where inspection is not rigid, the frequency is still greater. Sidney Martin[1] states that 10 to 20 per cent. of all cows are tubercular, and quotes statistics from Copenhagen showing a percentage of 17·7 for oxen and cows and 0·2 for calves; from Berlin a percentage of 15·1 and 0·06 respectively; while for swine the percentage was 15·8 in Copenhagen and 1·55 in Berlin.

Bang[2] also gives numerous statistics to show not only the great frequency of tuberculosis in cattle in general, but also the great variability in different countries and in different parts of the same country.

Fleming[3] gives a map which demonstrates for Baden-Baden a close relation between the prevalence of tuberculosis in cattle and the frequency of tubercular disease in the population.

A strong presumption exists against milk. For cattle, at any rate, the infectivity of the milk of tubercular cattle has been conclusively proved by experiment.

Hirschberger[4] took twenty tubercular cows, removed the udders with great care, and tested the milk obtained from them by inoculation into other animals.

The experiments[5] yielded positive results in 11 (*i.e.*, in 55 per cent.) and negative in 9 (*i.e.*, in 45 per cent.). The percentage of successful inoculations increased the more highly tubercular the animal was. Thus—

5 very tuberculous cows yielded 4 positive results and 1 negative.
6 moderately tuberculous cows yielded 4 positive results and 2 negative.
9 cows, in which the tubercle was limited to the lungs only, yielded 3 positive results and 6 negative.

In only one of the 20 cases was tubercle discovered in the udder itself, so that these observations prove that the milk of cows suffering from tubercle in any part of the body is risky.

Tubercular disease of the udder itself greatly increases the risk. It is fortunately rare, but possibly not so rare as is often asserted. Easton[6] states that at the present time it does not exist in more than 0·5 per cent. of cows in the London district. Tubercular disease of the udder may be a primary affection, *i.e.*, tubercle may exist in the udder and nowhere else.

Not only has the milk been proved to be infective, but the bacilli have been actually demonstrated in it, and in the butter and cream obtained from it.

It has been also shown that the infectivity of the milk is largely diminished by the practice in dairies of mixing the milk of many cattle together before distribution, the infection being very diluted and diminished in proportion; and, lastly, that the infectivity may be destroyed completely by boiling the milk for a few minutes, or even raising it for a somewhat longer time to a temperature just short of the boiling point.

The proof, then, of the infectivity of the milk of tubercular cattle for cattle is complete.

The evidences of the infectivity of the milk of tubercular cattle for man is less complete and satisfactory, but the presumption is strong.

1. Tubercle is very rare in the first few months of life, but becomes frequent after the first year, and continues prevalent for the next two or three years, after which it becomes less frequent again. During these years it is that cows' milk enters so largely into the diet.

2. The intestinal glands are a very common seat of primary tuberculosis in small children, and they must have become infected through the food.

[1] Clifford Allbutt, *System of Medicine*, ii. 32. [2] Tuberc. Congr., Paris, 1898.
[3] *Veterin. Med.* [4] *Deutsch Arch. f. klin. Med.*, vol. xliv. *Virch. Jahrb.*, 1890, i. 545.
[5] Bollinger, *Thiermed. Rundschau*, 1888, p. 272. [6] Tuberc. Congr., Lond., 1901.

3. Mesenteric tuberculosis, or tabes mesenterica, in children has not diminished at all, or in anything like the same degree, as phthisis has in recent years.

Direct proof of the infection of an infant by the milk of a tubercular cow is very difficult to obtain, for tubercle is of slow growth, and before the lesion has sufficiently developed to be recognised, all chance of tracing the infection to its source in a particular cow has usually been lost.

A few cases, however, which appear to be conclusive, are recorded.

A healthy child,[1] born of healthy parents, was fed exclusively upon the milk yielded by a particular cow ; the child died at the age of four months with tubercular mesenteric glands. The cow was slaughtered, and though supposed to be perfectly healthy, was found to be tubercular, and tubercle bacilli were demonstrated in its milk. Other cases are referred to by Ravenel.[2]

As all risk of infection by milk can be removed by boiling, this seems a simple and obvious thing to do, especially as tubercle bacilli are not the only infective organisms which are conveyed by milk. It is difficult to overcome prejudice, but the taste for uncooked milk is acquired, and children who have never had anything but boiled milk do not like it raw.

It has been asserted that milk is deprived of some of its nutritive value by boiling. This is an assertion which rests upon no evidence, and what evidence there is, is directly opposed to it.

Flesh.—The muscles are not a favourite seat of development of the tubercle bacilli. It is rare to find tubercular lesions in the muscles except in acute general tuberculosis, and not often then. Still the muscles of tubercular cattle, and even the expressed juice of the muscles, have been shown by experiment to be infective to cattle.

Meat, however, is rarely eaten raw in this country, and cooking greatly diminishes its infectivity, though it does not completely sterilise it.

The risk, therefore, in the case of meat is comparatively small.

In what has been stated, the practical identity of bovine and human tuberculosis, and the transmissibility of tubercle from cattle to man, have been assumed.

That tubercle in animals presented certain differences, both pathological and physiological, from that in man has been long known, but not regarded as of fundamental importance, or more than would admit of easy explanation on the analogy of other infective organisms.

It was, therefore, with great surprise that Koch's statement was received, that bovine and human tubercle were different diseases, and not communicable the one to the other ; and, as a practical conclusion, that the prevention of tubercle was to be looked for in general improvement in the sanitary condition of man rather than in the purification of dairies and in the inspection of cattle.

Koch's[3] position appears to be this :—

(1) The bacilli of bovine tuberculosis are much more virulent to cattle and the domestic animals than those of human tubercle.

(2) The difference is so marked as to be relied upon as a means of distinction between the two forms.

(3) If bovine tuberculosis were the common source of human tuberculosis, intestinal tuberculosis should be common in man, and not rare as it actually is.

To each of these propositions strong objections can be urged.

(1) Granted the low virulence of human tuberculosis for cattle, it does not follow that the converse is true, viz., that bovine tuberculosis has a low virulence for man. The probabilities are to the contrary, for the bacilli of bovine tuberculosis being so dangerous to such diverse species as the rabbit, horse, dog, pig, and sheep, indeed to almost every quadruped upon which

[1] Demme, *Schmidt's Jahrb.* for 1888 ; *Wien Med. Bl.*, x. 49, xi. 6.
[2] Tuberc. Congr., Lond., 1901.
[3] M'Fadyean, *Lancet*, Aug. 3, 1901.

they have been tried, it is in the highest degree probable that they are also dangerous to man; for all the bacteria which are pathogenic to all the domesticated animals are also pathogenic to man. Indeed, it may even be the case that bovine tuberculosis is especially virulent to man, as Ravenel maintains.

At any rate the *possibility of direct infection* of man from bovine tuberculosis is proved by certain cases of accidental inoculation, to which reference will be found in Ravenel's[1] communication.

(2) It does not appear to be yet proved that the difference in virulence between the two forms is so marked a characteristic as to be a reliable means of distinction between them, for there are great differences in the virulence of tubercle bacilli even in animals of the same species.

(3) Koch's third proposition is the most important. This states that if human tuberculosis had its usual origin in infected milk or flesh, primary intestinal tuberculosis would be common and not rare, as it actually is.

The statistics, however, on this point vary greatly. Thus, Still's figures, derived from the statistics of the Children's Hospital in Great Ormond Street, London, place the percentage of primary alimentary infection in tubercular children at 29·1; and Shennan, for the Royal Hospital for Children in Edinburgh, at 28·1.

It is quite true that tubercular ulceration of the intestines in children is rare compared with the frequency of tubercular diseases of the mesenteric glands, but this is to be explained by what appears to be the fact, that the bacilli can pass through the mucous membrane of the intestines without producing local lesions, just as they are apparently able to do through the mucous membrane of the bronchi, for the bronchial glands are often found tubercular without any tubercular lesion being discoverable in the air-tubes.

Koch's statements, made with so much conviction and authority, necessitate the complete re-examination and re-investigation of the whole question of the relation between human and bovine tuberculosis, and judgment must be suspended until the various commissions of inquiry appointed in different countries have produced their reports.

The complications are still further increased by the existence of many other acid-resisting bacilli, closely resembling, but distinct from, the tubercle bacillus, and this robs of much of their importance many of the statements made as to the existence of tubercle bacilli in milk, butter, or cream.

A good *resumé* of this part of the subject was given by Dr. Moeller in a paper read before the Tuberculosis Congress in London, 1901.

The great characteristic of the tubercle bacillus by which it has been hitherto recognised, viz., its resistance to acids and alcohol, is now known to be a characteristic, not of the tubercle bacillus alone, but of a group of bacilli of which the tubercle bacillus is only one. This property is not even essential to the tubercle bacillus. It depends upon a peculiar fatty and waxy envelope with which the adult tubercle bacillus is surrounded, but which the young tubercle bacillus does not possess. Moreover, the substance of which the envelope is formed may be chemically extracted and removed and the tubercle bacillus still remain alive and retain its specific properties.

Other acid-resisting bacilli resembling those of tubercle are the *lepra-bacillus*, the *smegma bacillus*, the organism of *avian tuberculosis*, the *butter bacillus*, the *manure bacillus*, found in the fæces of animals, and the *grass bacillus*, or *Timothy bacillus* (called so because found in the Timothy grass, *Phleum pratense*). All these resemble the tubercle bacillus closely.

The grass bacillus and the manure bacillus are the most important, but from them both the tubercle bacillus can be distinguished by its exceedingly slow growth on cultivation, the former forming colonies in twenty-four hours or so and at room temperatures, the latter only after several days, and at incubator temperatures.

These pseudo-tubercle bacilli have been also found in the sputum from the lungs of man, in the mucus of the nose and pharynx, on the tongue, in the sordes, on the teeth, and in the tonsils.

The confusion is rendered still greater by the fact that all these acid-resisting bacilli produce tubercle-like lesions in the animals experimented on, though only under special conditions and in a limited number of cases.

[1] *Loc. cit.*

It is thus evident that great as has been the advance made in the knowledge of tubercle in the last twenty years, we stand still only on the edge of the subject with an ever-widening field of investigation opening out before us.

If it be right to suspend judgment for the present as to the absolute identity of human and bovine tuberculosis, it will surely be wrong, while waiting for further knowledge, to suspend the measures now being taken to diminish the risks of infection, these measures being the logical conclusion from the facts so far as known at present. It will be well, therefore, to continue, for the present at any rate, to legislate for the purification of dairies and slaughter-houses, and the elimination of diseased cattle.

In respect of milk, it is surely wise to err on the side of caution, and, as boiling will destroy other infective organisms beside tubercle bacilli, to continue to boil the milk, especially as there is no evidence whatever to show that boiling in any way impairs its nutritive value.

Mode of Infection.—In animals it has been experimentally proved that tubercle may be conveyed to healthy animals by inoculation, inhalation, and feeding. In man the same modes are possible, though not all equally likely.

1. *Inoculation.*—Of recent years many conclusive cases of the inoculation of tubercle have been recorded in man, usually upon the hands. Many of the cases were treated surgically and cured, but others became phthisical after a time.

Thus the case is recorded[1] of a nurse who cut her finger in cleaning the broken spitoon used by a phthisical patient. The wound became tubercular and spread to the tendons, sheath of the muscles, and glands. Bacilli were demonstrated in the tissues. The patient was operated on and cured.

A similar case is quoted by Deneke[2] in a child, who cut his head with the edge of a utensil in which his phthisical mother spat. The wound became tubercular and so did the glands, and the child died four months later.

Steinthal[3] also gives the case of a wife who acquired a tubercular skin affection of the hand as the result of washing the linen of her phthisical husband.

It is probable that many of the *post-mortem* warts are nodules of tubercular infection. Karg[4] records the case of a *post-mortem* attendant who died of phthisis, the result, it appeared, of such an inoculation.

Another interesting group of cases[5] is that in which tuberculosis developed in circumcision wounds, the wound having been sucked at the time of operation by the operator, who was phthisical.

The possibility of direct inoculation in man may be regarded as established, but this mode of conveyance is obviously of rare occurrence, and if we may judge from tuberculosis of the skin, phthisis is but a rare result of such inoculation.

2. *Feeding.*—On the analogy of cattle we may fairly conclude that there is considerable risk of infection to the child if it be suckled by a tubercular mother, although the risks must be somewhat less than in cattle, inasmuch as tubercular mastitis in woman is extremely rare.

Another source of risk to the child is the practice, which is not uncommon among mothers, of chewing portions of the child's food themselves before putting it into the infant's mouth. With a phthisical mother, the chance of infecting the food in this way would be considerable.

[1] *Cf.* Heron on *Communication of Phthisis*, p. 25. Other cases in appendix by Koch, Gottstein, *Fortsch. d. Med.*, 1886, No. 8.

[2] *D. med. Woch.*, Mar. 27, 1890.

[3] *D. med. Woch.*, 1888, No. 10.

[4] *Centralbl. f. Chirurgie*, 1885, Aug. 5.

[5] Eve, *Lancet*, 1888, i. p. 28. Elsenberg, *D. med. Ztg.*, 1886, p. 358. Lehmann, *D. med. Woch.*, 1886, Nos. 9–13.

Tubercular mothers, therefore, should not suckle their infants, and should be warned of the need of care lest the infection be conveyed from their own mouths.

What applies to the mother applies equally to a wet nurse, and no woman who is actually phthisical, or is suspected of being so, should ever be employed as a wet nurse, for there are many instances recorded in which a child born of healthy parents, and whose only food was obtained from the breast of a phthisical wet nurse, developed tuberculosis and died of it.

If cow's milk be used as the substitute for mother's milk, whatever risk of infection from it there may be is removed by boiling, and boiling should never be omitted.

3. *Inhalation.*—In animals inhalation is the most certain way, after inoculation, of conveying tuberculosis, and, *a priori*, the presumption is strongly in favour of inhalation being also the most common mode of infection in man, considering the ubiquity of the tubercle germ and its free dissemination with the dust.

There is, however, no evidence to show that there is in man anything which corresponds with the acute tubercular broncho-pneumonia experimentally produced by inhalation in animals, probably because the infective material is not introduced into the lungs of man in sufficient amount. When this form of acute tuberculosis of the lung is met with in the adult or in the child, it is generally to be traced to the actual discharge into the air-passages of tubercular material, from a caseous gland or other tubercular focus communicating with the air-passages.

In most cases, there is good reason to believe that the bacilli introduced by the air, whether inhaled or swallowed, locate themselves in the lymphatic glands, and that the mischief in the lungs is secondary to that in the glands.

Direct Infection from Man to Man.—In this connection the question naturally arises, Whether phthisis is catching—that is to say, whether it is communicable from the sick to the healthy?

The question in the case of phthisis narrows itself to this. What evidence have we that phthisis may be conveyed to a healthy person by the inhalation of the breath of a phthisical patient? The bacilli have been found,[1] it is true, in the air expired, but careful observation has shown that they do not exist in the air as such, but in the particles of sputum thrown into it by coughing.

Direct infection, therefore, by means of the breath is a possibility, but we must enquire whether such a mode of infection is supported or proved by clinical evidence. It is fair to say at the outset that if phthisis were eminently contagious, the proof of it ought to be overwhelming, considering the frequency of the disease; and yet we see that great doubt exists on this point among those most competent to judge, and that some who at one time believed in its contagiousness abandoned that opinion. Of these Louis, Portal, and Laennec are the most notable instances. The question is one which must be settled by evidence and not by theory.

If phthisis were a contagious malady we should expect to find the clearest proof of it among those who are placed in close relation with the sick, *e.g.*, among married couples, among nurses and doctors, and among inmates of the same house or institution. The problem is very complicated, for, quite apart from the question of contagion, there is clear evidence that external conditions, such as the place and mode of living, exercise an important influence which it is very difficult to eliminate.

[1] Ransom, *Proc. of Roy. Soc.*, vol. xxiv.

I. *Infection from one married partner to the other.*—In considering the evidence offered in favour of direct infection as between man and wife, one cannot fail to be struck with the rarity of the cases. Thus Wilson Fox,[1] out of a large experience of twenty-seven years' duration, can only quote two instances, and in one of these the second partner was not seen till five years after the death of the first. Hermann Weber,[2] again, in his oft-quoted paper, records 9 cases only out of an even longer and larger experience. For myself, although I have been on the look-out for such cases, I have only met with one instance in the course of twenty-five years, and that would hardly bear very critical investigation.

In the Collective Investigation Records many of the contributors say that they have seen but one case in many years, for instance in forty-five, forty, thirty-six years and so on. Even on the general question, in that report the answers in the negative bear to the answers in the affirmative the proportion of about 3 to 1, while even among the affirmative answers many are mere statements of opinion without facts or reasons given.

Some writers, however, place the frequency much higher; thus Leudet[3] out of 112 cases met with 7 instances in which both married partners were phthisical, *i.e.*, 6 per cent.; Wahl Essen's[4] series yielded a percentage of 8, and Osler's[5] also of nearly 6.

Schryder gives the following figures:—Of 844 cases of phthisis in married persons, the man only was phthisical in 445, the woman only in 367, and in 32 cases, *i.e.*, 3·8 per cent., were both phthisical; in some of these instances both were phthisical before marriage.

Phthisis is so common a disease that the chances cannot be inconsiderable that a man, born to die of phthisis, whether he married or remained single, should wed with a woman also born to die of phthisis whatever her career in life. Longstaff[6] has calculated these chances and found them to be approximately as much as 3 per cent., *i.e.*, that among 100 married couples the chances are that in three both husband and wife will die of phthisis without any other cause than accidental association. This percentage must then be deducted from the total given for such cases, and thus even the outside percentages of 6 per cent. or 8 per cent. just quoted would be reduced to 3 or 5, or if the common experience be taken the percentage would fall to zero. Statistics therefore seem to prove the risk of direct infection between man and wife to be very small, for taking even the outside estimates, the chances are at least 12 to 1 that where the one partner is phthisical the other will escape, and if not, at least 20 to 1 that the disease will have been acquired in some other way. Again, it is strange on the assumption of direct infection that there should be such a difference between the sexes as the figures show, for it seems to be twice as frequent for the husband to infect the wife as for the converse, viz., for the wife to infect the husband, and as the general liability to phthisis is the same for both sexes, this would point to some special cause other than infection.

The common class of case quoted is that in which a husband dying of phthisis is nursed by his wife, who after his death becomes also phthisical; but in many of these cases the connection is somewhat strained, for the intervals between the husband's death and the appearance of phthisis in the wife is often considerable, and may be months or even years.

The most remarkable cases of all are those in which the same phthisical husband loses more than one wife of phthisis, in one instance four wives (Weber),[7] in three other instances three, and in nine other two.

On the other side must be placed those much more frequent instances in which one of a married pair is phthisical, and the other, though living in the

[1] *Dis. of Lungs*, p. 571.
[2] *Trans. Clin. Soc.*, vol. vii.
[3] *Bull. Acad. Med.*, 1885, No. 15.
[4] *D. med. Woch.*, 1885, p. 4.
[5] *Medicine, l.c.*
[6] Appendix to Collective Investigation Records.
[7] Clay, *Étude de la contag. de la Phthisie*, Paris, 1879. *Cf.* note in Wilson Fox, p. 571.

closest association, never becomes phthisical at all. Portal records two cases in which a man married in the one instance two and in the other three wives, all of whom died of phthisis, and yet the man in each case escaped. On the whole, taking the evidence as it stands, the risk of direct infection as between man and wife must be very small.

The transmission of infection "per coitum" has been suggested, but it is unproved in man, and even in animals the cases quoted seem hardly conclusive. Thus Zippelin[1] quotes an instance in which a young tubercular bull infected ten cows, and Haarstuck another in which sixty cows were infected, but it does not follow that the other possible modes of infection were excluded.

If direct infection be difficult to prove between man and wife, it must be still more difficult to establish in the members of a family.

Ogston[2] records the case of a family of ten persons without hereditary taint. A son became phthisical abroad, and was nursed at home by his two sisters, while a brother slept with him. These three and the patient died of phthisis, the parents and the four surviving brothers remaining healthy.

As the type of this class the following case may also be quoted. In a very healthy family a brother suffering from phthisis was nursed by his three sisters, all of whom ultimately became phthisical, while another brother who was not at home remained healthy.

Cases of this kind are often also quoted as evidence of the effect of local influences, *i.e.*, to prove that the infection attaches rather to places than persons, but it is clear that they cannot do duty for both purposes.

II. *Infection among the attendants on the sick.*—In this respect the evidence that is forthcoming is strongly opposed to the theory of direct infection.

The combined reports of the two great London Chest Hospitals,[3] at Brompton and Victoria Park, show that of all the officers, staff, officials, servants and nurses working in them for a number of years (twenty to thirty-six), out of a total of 779 persons only 30 became phthisical, of whom 23 were known to have died, *i.e.*, 3·8 per cent. Owing to certain gaps in the returns, the percentage is really less than this, and the returns are still more unfavourable to the theory of direct infection. The percentage, however, as it stands is less than that of the selected lives of insurance offices.

A recent report from the Friedrichshain hospital in Berlin shows the same results. Thus, out of 459 male nurses only 4 were phthisical, and 2 were phthisical before entering the service; and out of 339 female nurses only 2 became phthisical, *i.e.*, a rate of only 0·5 per cent.

III. The prevalence of *phthisis in institutions* will be shown later to depend chiefly upon sanitary conditions, and to be greatly reduced where the sanitary conditions have been made good.

Lastly, the increase of phthisis during recent years in Australia and New Zealand, which has been referred to infection from the phthisical patients sent there from other countries, is probably much more intimately connected with the change of habits of life in the population, the people being no longer, as at first, sparsely scattered over the country, but now largely massed in towns.

The facts, therefore, seem to point to these conclusions—

1. That, though a communicable disease, phthisis is either not at all, or else in a very limited sense, contagious, and that the risk of direct infection from the sick to the healthy is very small.

2. That inhalation is probably the common mode of infection, and that indirectly through infected dust, with which the air is laden. In this respect must be borne in mind (1) the ubiquity of the bacillus; (2) the probable exposure of all to infection; and (3) the great powers of resistance of the healthy body.

[1] *Cf.* Johne, *Gesch. d. Tuberc.*

[2] *Brit. Med. Jour.*, 1884, ii. 11.

[3] Th. Williams, *Brit. Med. Jour.*, 1882, Sept. 30. Cotton, *Lancet*, 1887, ii. 550. Andrew, Lumleian Lect., 1884, *Lancet*.

[4] Fürbringer, *Lancet*, July 5, 1890.

Conditions Influencing Infection.—Considering the wide diffusion of the tubercle bacillus, it is probable that every one has been exposed at some time, and perhaps very often, to the risk of infection, yet not more than about 15 per cent. of the population fall victims to phthisis. Hence it follows that about 85 per cent. offer more or less successful resistance to its attack. In some, at least 50 per cent., the resistance is complete, and in the remaining 35 per cent. sufficient.

Health is not a fixed quantity, but consists in a constant adjusting of the balance between the forces which tend to impair it, and those which maintain it, in such a way that the resistance offered by the body is always greater than the attack made upon it.

Neither of these quantities being fixed, the equation for health would be

(R $\propto >$ A) Resistance varies with, but remains greater than, Attack.

On the other hand, the equation for disease would be

(R $<$ A) Resistance is less than Attack.

This last equation may be arrived at in different ways, by increasing the value of the attack, or by reducing the value of the resistance or both.

Applying these considerations to phthisis, the risk to any individual of becoming tubercular may be increased either by increase in the power of the attack of the germ upon the body, or by a diminution in the power of the body to resist it. External conditions can only affect phthisis by acting in these two ways.

A. The power of attack of the bacillus can be increased so far as we can judge in only two ways, *i.e.*, by an increase either in their virulence or in their number.

Though it is possible that the virulence of the tubercle bacillus may vary under certain conditions, there are, I believe, no facts which prove this at present.

That the risk varies[1] directly with the number of bacilli introduced into the body has been experimentally shown; in other words, concentrated infection is more dangerous than dilute. Still, among individuals placed under similar conditions the susceptibility varies greatly.

It is evident, therefore, that the varying susceptibility of individuals to tuberculosis depends rather upon variations in the resistance offered by them, than in the violence of the attack made upon them.

B. The natural resistance of the human body is evidently considerable, for it is only about 15 per cent. of the total mortality that is due to phthisis. Of the remaining 85 per cent., some have resisted the attack completely, so that no tubercular lesion is found in the body at the time of death; in the others tubercular lesions are found, but they are localised or "healed."

The frequency with which the lesions of "healed" tuberculosis are found in the lungs is differently estimated by different writers, but it is recognised now as being much greater than used formerly to be thought. This is chiefly due to the fact that many of the lesions which used to be placed in other categories are now proved, or for good reasons believed, to be tubercular.

Thus Heitler[2] puts the percentage of healed tuberculosis at 4·7 per cent. He excludes all simple fibrous cavities in the lung entirely from his figures. Coupland's and Fowler's[3] statistics yield a percentage of about 9. Recent observations made *ad hoc*, and with the present more

[1] Bollinger, *Münch. Woch.*, 1889, No. 43.
[2] *Virch. Jahrb.*, 1880.
[3] *Brit. Med. Jour.*, 1891, Oct. 31.

liberal interpretation of tubercular lesions, place the percentage very much higher, at the average of about 30 :—

Coats,[1] 23 per cent. ; Bollinger,[2] 27 per cent. ; Harris,[3] 38 per cent. At the same time Sidney Martin [4] puts it at 9·4 per cent.

Accepting these figures, it is clear that the resistance is perfect in at least 50 per cent., on the assumption, which I think we may justly make, that everyone has been exposed to infection ; that it is inadequate in from 15 to 20 per cent., and that in the remaining 35 to 30 per cent. the resistance, though not sufficient to prevent the bacillus gaining a lodgment in the body, is still sufficient to localise it, and, more or less successfully, prevent its extension.

Whether natural immunity exists in man is not known, and even in animals it is doubtful, for if they are free in the natural state, they become readily susceptible under artificial conditions.

The conferring of immunity by artificial means appears possible, but has not yet been achieved.

Protective inoculation with the products of tubercular action gives so far little promise of success. Otherwise, it is difficult to understand why, when tubercle is producing its own antitoxin, relapses of tuberculosis should so often occur, and why, indeed, tuberculosis has not stamped itself out long ago by natural selection. The actual results obtained from Koch's tuberculin have disappointed the hopes held out.

The exact mode in which the tubercle bacillus, after it has gained access to the body, is limited and controlled in its action is no doubt the same as with other pathogenic germs.

The resistance of the body may be reduced by causes (*a*) which have their origin within the body itself, or (*b*) which act upon the body from without.

(*a*) The causes which have their origin within the body may be natural or acquired, *i.e.*, the individual may possess a natural resistance below the average, or, possessing a natural average resistance, may have it reduced by accidental causes. To the first group would be referred the effects of race, inheritance, constitution, age and sex ; to the latter, the causes of feeble health and general disease, or previous disease of the lungs themselves.

(*b*) Among the causes which act upon the body from without would be ranged, the effects of climate, density of population, habits and conditions of life, unsanitary occupations, and insufficient food.

The part which impaired health or previous disease plays in the causation of phthisis will be most naturally dealt with when considering the relation which exists between phthisis and other diseases, and will be best deferred until the influence of the other conditions enumerated has been discussed.

Race.—No race is immune. Though differences in race susceptibility exist in different countries, they depend upon habits of life and other conditions rather than upon peculiarities of race.

For instance, in India the white races suffer more than the native, while in Egypt it is the reverse. Phthisis is the great scourge of the negro transferred from his own habitat to a temperate or cold climate, but even without change of climate the same change in susceptibility occurs, *e.g.*, the West-Indian negro,[5] who is not specially susceptible to phthisis at home, becomes so when transferred to the Gold Coast, though his near relations there, the natives of the Gold Coast, are almost free from it. Again, in America phthisis is rapidly increasing among the Indian races, among whom it was said to be unknown. This is probably due to the changes in the habits of life which the colonisation of the country brings with it.

One remarkable fact in the race-history of phthisis is the comparative freedom of the Jews all the world over, and it is tempting to connect this with the ordinances of their religion, which

[1] *Brit. Med. Jour.*, 1891, Oct. 31.
[2] *D. Arch. f. klin. Med.*, vol. x.
[3] *Brit. Med. Jour.*, 1889, Dec. 21.
[4] *Ibid.*, 1891, Oct. 31.
[5] Andrew's Lumleian Lect., *Lancet*, 1884.

inculcate thorough cleansing of their homes at least once in the year, and constant supervision of the food supplies. It is stated, further, that this exemption, while still existing among the strict Jews, is being lost among the non-conforming Jews.[1]

MacLaurin[2] stated that in N.S. Wales, in a population of 4000 persons, the mortality from phthisis among the Jews was as 1 to 13 among the Christians.

Climate.—Phthisis is found all the world over under every condition of climate.

It prevails most where the population is densest, and that not only in absolute but in relative amount.

The mortality is much the same in all large towns.

For instance, the rate is about 3·6 to 3·8 per 1000 persons living, in towns so differently placed as London, Boston, St. Louis, Charleston, Copenhagen, Malta.[3]

Even where differences exist they are often too great to be explained by the differences in climate simply.

In 1886 the rates were for London 2·02, for Paris 4·7, for Vienna 6·7, for Buda-Pesth 7·1. Even in adjacent places the rate varies greatly; thus it is rare in the inland parts of Asia Minor, but common on the coast. It is four times as frequent in Cairo as in the other parts of Egypt. It is rare in the islands off the west coast of Scotland, and yet common on the mainland. It is three times more frequent in Glasgow than in Edinburgh.

Lastly, it is increasing in places which used to be free from it.

Thus it is becoming as frequent in the Australian towns as in the towns of Europe, and it is very prevalent among the Indians at the foot of the Rocky Mountains, in what is, perhaps, the finest climate in the world.

The strongest argument of all against the direct influence of climate lies in the different rate of mortality of the two sexes even in the same place.

Rate per cent. living.[4]	Males.	Females.	Excess.
Cambridge, ...	5·7	3·95	1·75 males.
Bath, ...	5·4	2·55	1·85 ,,
Salisbury, ...	4·1	3·05	1·05 ,,
Bootle, ...	2·25	5·55	3·3 females.
Buckingham, ...	2·75	4·55	1·8 ,,
Sevenoaks, ...	2·9	4·55	1·65 ,,

These various arguments show that climate, as such, can have but little real determining influence in the development of phthisis.

The three most important factors in the production of climate are temperature, altitude, and soil, and the effect of each may be considered separately.

Temperature.—Phthisis abounds in all temperate climes, but it is quite as common in the Tropics, and is not rare even in the Arctic regions.

Thus it is frequent in Ceylon, Mauritius, China, and the Gulf of Mexico, but so it is too among the Esquimaux in Greenland and among residents at Archangel and Alaska.

Again, mildness of climate has little effect.

It is rare in Newfoundland and common in the Ionian Islands; it is equally common in London and Boston; it is almost unknown in Iceland and the Faroe Islands, but so it is also in Senegambia and on the Gold Coast.

On the whole there seems to be reason to believe that phthisis runs a more rapid course in tropical climates, but it is certain that great variability of climate has a predisposing influence owing to the prevalence of catarrh.

[1] Heron, *l.c.*, p. 96.
[2] Internat. Med. Congr. at Melbourne.
[3] Lebert, *Brustkr.*, 476.
[4] Ransom, *Brit. Med. Journ.*, March 8, 1890.

Altitude.—There is a general opinion that altitude is in some respects antagonistic to phthisis. The maps show that high plateaux and mountains are comparatively free, but they are also the most sparsely populated regions, and there are notable exceptions, for instance, the Kirghi Steppes, which lie below the sea level.

Schrötter's[1] observations show for Switzerland the diminution in the mortality from phthisis as the elevation increases.

6–1200 feet, . .	112 per 1000.	27–3600 feet, . .	92 per 1000.
12–2100 ,, . .	105 ,,	3600 and over . .	71 ,,
21–2700 ,, . .	106 ,,		

Muller's[2] observations, which show, though not so conclusively, the same thing, prove also that the advantage gained from elevation may be easily overcome by other unfavourable influences, *e.g.*, indoor work and sedentary occupations.

In Europe great altitude is rarely compatible with a good climate. For this combination we must go to the western parts of the American continent, *e.g.*, Puebla, Quito, Mexico, Bogota, etc., where, in spite of many sanitary defects, phthisis is almost unknown.

Moisture.—Mere humidity of the air is of no influence one way or the other, for phthisis is common at Lima where the air is very moist, and in Egypt where it is dry; while in Devonshire, which is a very damp and rainy county, there is much phthisis.

Soil.—Bowditch[3] in America, and Buchanan[4] in this country, have shown that phthisis is unduly prevalent upon a moist impenetrable soil, and that its prevalence can be diminished by drainage.

Milroy[5] quotes in support of this view the case of Leith and Edinburgh, on a dry soil with a mortality of 2·98 per cent.; and Glasgow and Greenock, a wet soil, with a mortality of 4·0 per cent. As against the evidence in support of their case must be quoted the instances in which either no such improvement followed complete drainage, as at Berlin, or those in which an actual increase occurred, as at Dantzic, and at Ashby-de-la-Zouche, where the increase after drainage was actually 19 per cent.

The influence of a damp subsoil will be best considered in another place, in connection with that of locality and unsanitary dwellings.

There has long been supposed to be an antagonism between ague and phthisis, phthisis being almost absent from places where ague abounds, and *vice versâ*. Our present knowledge of the pathology of ague robs this opinion of all importance.

All these facts seem to show that climate, as such, has but little influence upon phthisis, except so far as it conduces, or not, to a healthy outdoor life, and that even the most favourable conditions of climate are easily counteracted by unfavourable conditions and habits of life.

Density of Population.—The maps, giving the geographical distribution of phthisis, show that it is most prevalent in the most thickly populated, and least in the most sparsely populated, parts of the world. This is not apparent only, and due possibly to fuller returns being forthcoming of the more populous places, but it is a real difference, and holds not only of the world at large, but also as between the town and the country, and even as between different parts of the same town.

[1] *Virch. Jahrb.*, 1889, i. 354.
[2] Hirsch, *Geogr. Path.*, p. 144.
[3] Med. Comm. Massachusetts Med. Soc., 1862.
[4] Tenth Rep. of Med. Off. of Privy Council for 1867; Seventh Rep. of Reg.-Gen. for Scotland.
[5] *Cf.* Hirsch, *loc. cit.*, p. 139.

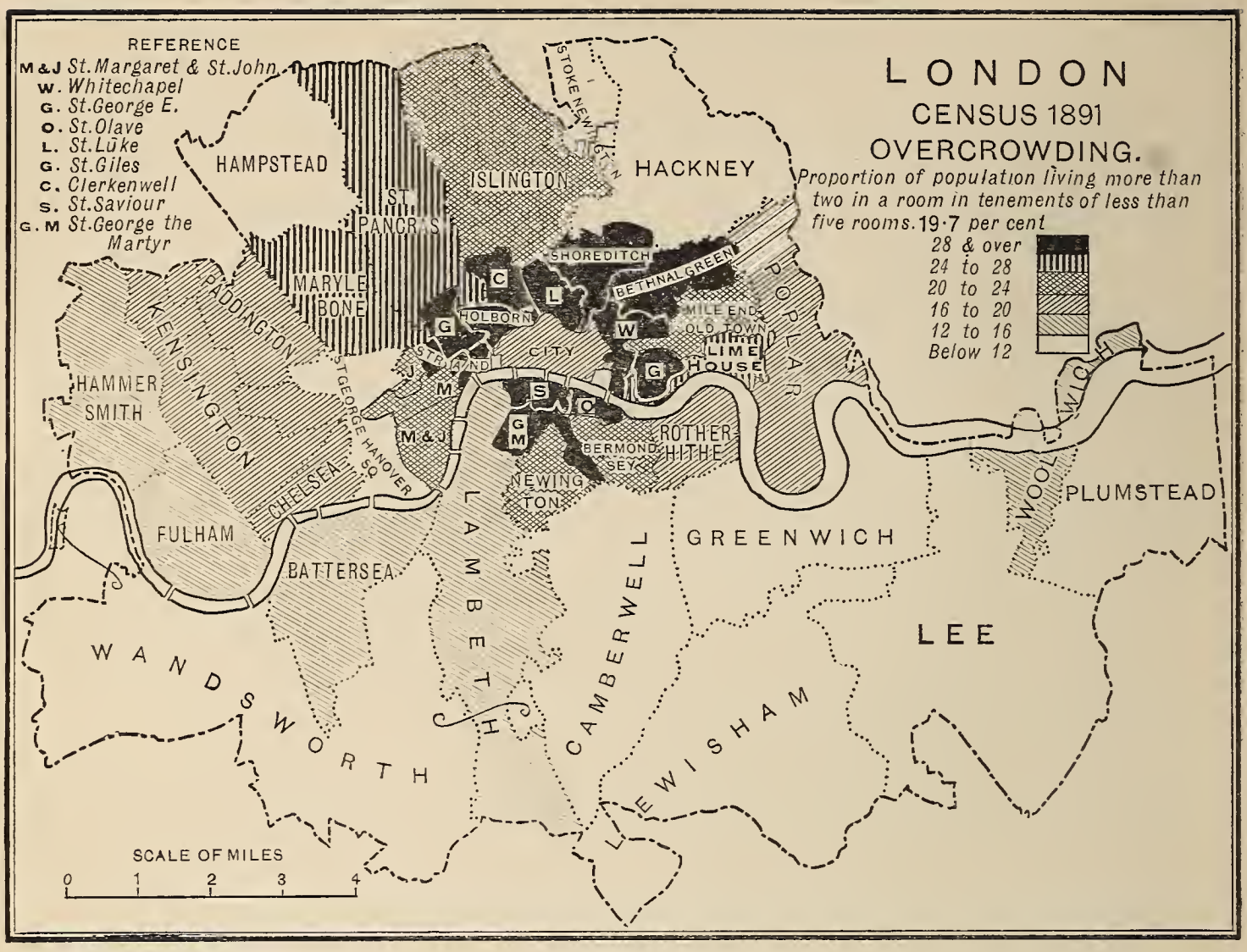


Fig. 121.

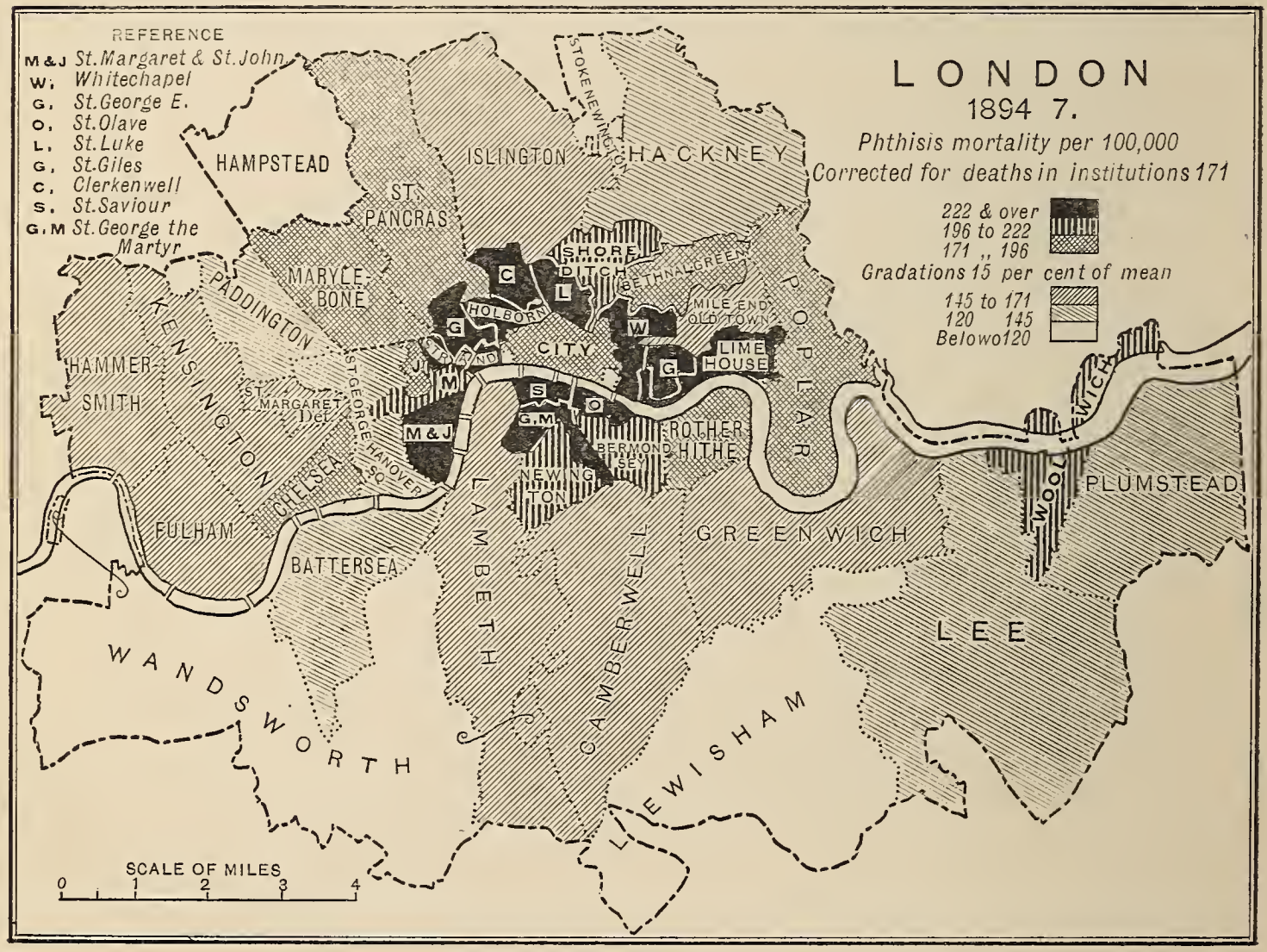


Fig. 122.

Maps to show the close relation between overcrowding and the mortality from phthisis. (Shirley Murphy—Tuberc. Congr., London, 1901.)

London, 1898, Mortality and overcrowding. (Shirley Murphy—Tuberc. Congr., London, 1901.)

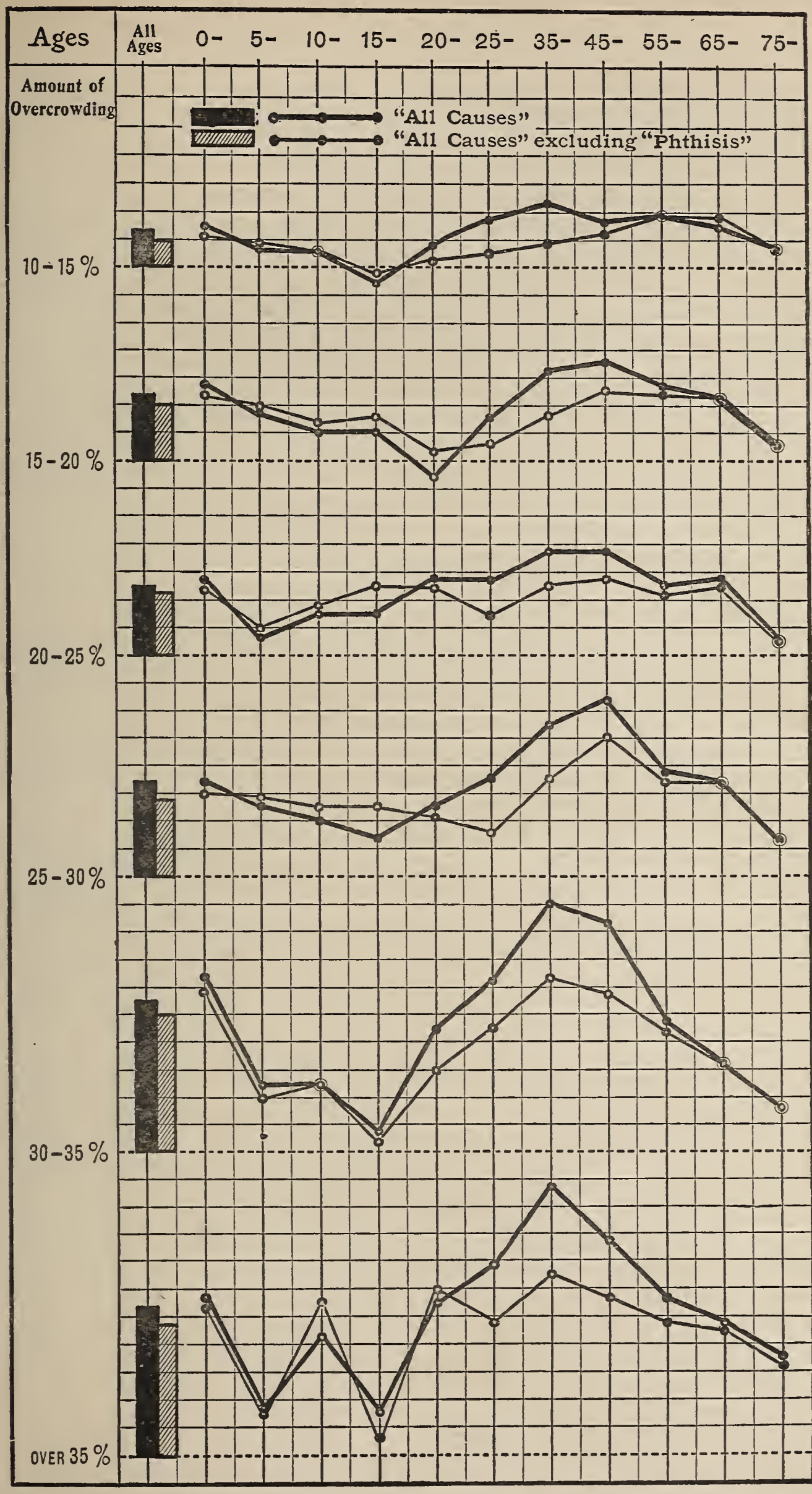


Fig. 123.—Diagram showing comparative death-rates from "all causes" and from "all causes excluding phthisis" at certain age-periods in groups of London sanitary districts, arranged with respect to their condition as to overcrowding.

Thus Farr showed that the death-rate for twenty-five towns was 4·6, while that for seven country districts was 3·7, thus giving a difference as against the towns of ·9, or a ratio of 100 to 80. The ratio for Bavaria is given as 100 to 61 (Majer),[1] for Holland as 100 to 76, while the returns from thirty-five districts in Prussia work out at 100 to 90. The figures, therefore, though showing differences, all point to the same conclusion.

Farr also gives a table showing the same results, though arrived at in another way, viz., by estimating the density of population by the number of square yards to each person. Thus with 57 square yards each, the mortality per 1000 persons living was 4·78; with 78 square yards, 4·51; and with 217 square yards, 3·54.

Ransom[2] carried his investigations even into the crowded parts of the same town, and showed that in Manchester and Salford phthisis specially prevailed in the close courts and alleys, in the shut-in or blocked-up lanes, and, above all, in the houses built back to back. Smith has also shown that in the London Hospital 70 per cent. of the cases of phthisis came from the crowded houses of the neighbourhood.

Dr. Shirley Murphy has also shown for London, that in the five groups of sanitary areas, the more over-crowded have higher death-rates for phthisis, at each age period, than the less crowded.

It is, of course, impossible to separate the effects of mere density of population from the natural results of overcrowding, viz., unsanitary, ill-ventilated dwellings, insufficient food, unhealthy occupations, and the keen struggle for existence under anxious and unfavorable circumstances.

Sir Hugh Beevor[3] gives the results of a similar investigation, with instructive maps and diagram for London.

General Conditions of Life.—Phthisis is more frequent among the poor than the rich. It is of shorter duration, and shows a smaller percentage of cures.

Thus Guy,[4] quoted by Wilson Fox, gives the proportions as 16 for the upper classes, 20 for the tradespeople, and 30 for the artisans. In Geneva the mortality has been stated to be three times as great among the poor as among the rich.

Occupation.—The mere change from an outdoor to an indoor life greatly increases the phthisis-rate. The difference between the rates in town and country has been already stated, and that may be taken also as expressing the difference between industrial and agricultural occupations. Simon draws the following conclusions from his statistics. "In proportion as the male and female population are severally attracted to indoor branches of industry, in such proportion, other things being equal, their respective death-rates by lung diseases increase."

Simon and Greenhow[5] have also pointed out the great difference in the death-rate for phthisis in different districts, showing that they may vary from 13·4 to 44·5 (the female from 15·6 to 51·7 and the male from 22·9 to 58·8). This, they argue, points to the operation of local causes.

But besides this, the liability to phthisis is greatly affected by the nature of the occupation followed. It is especially the dusty trades that lead to phthisis; not that the dust as such is the cause of phthisis, but that it prepares the way for it by the changes it produces in the lung. All the dusts are not equally deleterious, the most mischievous being the metallic and then the mineral. In some trades phthisis is so common, *e.g.*, in knife grinding, millstone making, coal mining, that knife-grinders' phthisis, millstone-makers' phthisis, and miners' phthisis have come to be generally recognised terms. The connection of the prevalence of phthisis with the trade is conclusively shown, not only by comparing the different trades with each other, and both with the average incidence of phthisis in the district round, but also by the unequal incidence of

[1] Hirsch, *loc. cit.*, 148.
[2] Lect., *loc. cit.*
[3] *Lancet*, April 15, 1899.
[4] Marc d'Espine, quoted by Ransom.
[5] Surg.-Gen. Rep., 1858.

phthisis on those engaged in the particular trade, the increased liability falling on those only who are engaged in the dusty part of the work. The difference of employment thus explains the striking differences between the sexes often met with, the men or the women showing the excess according as they are employed in the dusty work or not, and both sexes only being equally affected when both perform the same duties. Lastly, the direct connection of the dusty work with phthisis has been proved by the diminished mortality coincident with sanitary improvements, such as wet grinding instead of dry, the wearing of respirators, or more perfect ventilation.

These questions have already been considered when treating of the dust diseases or pneumono-konioses, and will be further dealt with when the effect of previous lesions of the lungs upon the liability to phthisis is under consideration.

Unsanitary Surroundings.—The most striking evidence of the effect of unsanitary conditions upon the mortality from phthisis is shown in the case of large institutions, such as barracks, convents, and prisons.

Among the Troops.—In Canada the mortality from phthisis between the years 1830 to 1837 amounted to 23 per 1000, while for the corresponding ages in England and Wales it was only 7·7. The barracks were drained and ventilated, and between 1863 to 1872 the mortality fell to 9·5, and in the year 1874 it stood as low as 6·0.[1]

The general average of phthisis in the army used always to be in excess of that among the civil population. Thus in the report published in 1858, while the phthisis rate for the whole population was 6·3, it was for the cavalry 7·3, for the infantry 10·2, and for the guards 13·8. Great reduction in these rates has followed sanitary improvements in the barracks, but the rate for the army still remains above that for the general population.

Parkes[2] refers to the varying mortality in the army in different stations even in the best climates, and to the improvement which has taken place all round, attributing it entirely to the improved sanitary conditions of barracks. All military authorities agree that phthisis is rarer in the field than in the barracks, and that it is the great plague of an army in the time of peace.

Convents.—Cornet's[3] report on the nursing communities of Prussia shows that the death-rate from phthisis among them is more than three times that of the rest of the population, for while in the latter it was about 15 to 20 per cent. of the total mortality, among the nursing communities it reached nearly 63 per cent., and this, too, among selected lives, for no candidate was admitted to the order unless in good health at the time of application. The average age at death was only 36·27, so that the expectation of life of a young healthy person at the age of 25, which, under ordinary circumstances, would have been forty years on admission into the orders, fell to eighteen years; in other words, was not better than that of a woman at the age of 58.

Prisons.—Baly[4] in his report upon Millbank Prison showed that the death-rate from phthisis was nearly three times that for the general population, being 43 as compared with 13 per cent. Parkes[5] quotes the experience of two Austrian prisons, the one well ventilated, the other badly, and the death-rate from phthisis being in the former 7·9 and in the latter 51·4. Cless[6] quotes a prison in Würtemberg, in which, from 1850–9, the mortality from phthisis was 24 per cent. This was reduced by improved sanitation to 8 per cent. and was still three times as great as that of the general population. Similar statements are made with reference to harems and to overcrowded hospitals.

Facts such as these make it impossible to resist the conclusion that one of the most important determining factors in the development of phthisis is the living under unsanitary conditions in over-crowded, ill-ventilated houses, and this seems even to apply to individual houses.

Ransom[7] has drawn attention to the way in which phthisis seems to stick to certain houses, and quotes some statistics given him by Dr. Niven of Oldham. In the ten years, 1877 to 1887, 3001 deaths from phthisis took place in Oldham, most of them in the worst kind of

[1] Welch, Alexandrian Prize Essay on Lung Disease among Soldiers, 1872, p. 94.
[2] *Hygiene.* [3] *Ztschft.* f. *Hygiene*, vol. vi. p.
[4] *Med. Chir. Tr.*, xxvii. [5] *Hygiene.* [6] *Cf.* Hirsch.
[7] *L.c.*, p. 529. Tuberc. Infection Areas, Epidem. Soc., 1888.

house, and in 302, *i.e.*, just 10 per cent., more than one death from phthisis occurred in the same house. The chances of one house being twice affected accidentally were only 68, whereas 274 were so affected, and the chances of the same house being three times affected were only 7·6, whereas 24 were so affected.

Flick showed also that a house which had had one case of phthisis was likely to have another, and might have many in succession, and that tuberculosis of different forms often occurred in the same house.

Engelmann [1] gives an instance of a tenement in a flat which had been free from tubercle for eight years, and was then occupied by a tubercular patient for one year. In the course of the next twelve years at least 12 cases of tubercular disease were traceable to this source, tenant succeeding tenant, and the flat being never once painted nor thoroughly cleaned.

It is evident, therefore, that the sanitary condition under which persons live have an enormous influence upon their liability to phthisis, and it is difficult, as already stated, to make due allowance for this when dealing with the question of infection, for it is impossible to say whether the increased liability is due to more concentrated infection or to diminished resistance consequent on impaired health.

Phthisis mortality and its diminution in recent years.—The mortality from phthisis has to be considered in two relations—

1. In relation to the number of persons living at the different age-periods.

2. In relation to the mortality from all causes.

The two accompanying charts show this in diagrammatic form.

There is no doubt that the general mortality from phthisis has considerably diminished of recent years—and it is in accordance with the evidence to refer this largely to the improved sanitary conditions under which the people at large live.

Similar investigations made by Lawson for the army show a diminution of nearly 60 per cent. comparing the two periods, 1837–46 with 1880–84.

The influence of the conditions, under which the persons live, comes in to complicate very greatly on the one hand the question of the contagiousness of phthisis, and on the other that of inheritance, for it is now clear that many cases of family phthisis may be the result not of inherited predisposition but of acquired susceptibility.

The conditions of life seem to act in one of two ways—either by impairing the general health, and so diminishing the general resistance of the body, or by producing local lesions in the lungs, as certain occupations tend to do, and thus diminishing the local resistance of the lungs.

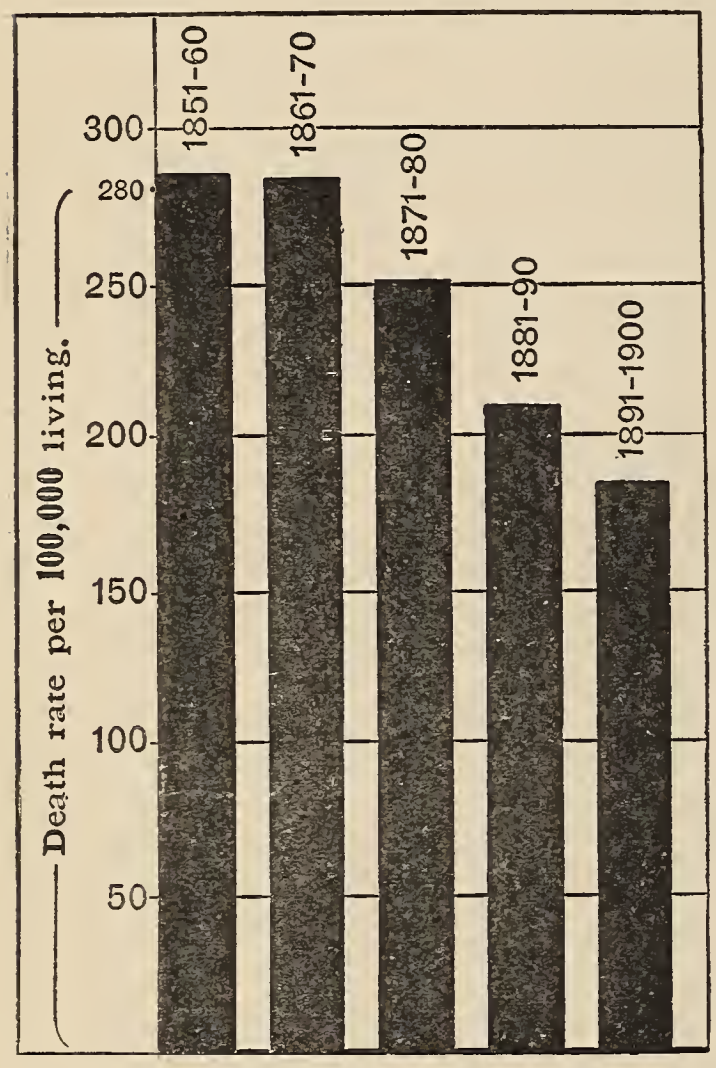


Fig. 124.

Diagram showing deaths from phthisis per 100,000 living in successive decennia (London, 1851–1900). (Shirley Murphy —Tuberc. Congr., London, 1901.)

[1] *Brit. Med. Jour.*, April 18, 1899.

That loss of general health is a real predisposing factor is evident from the occurrence of phthisis during convalescence from acute illness, for instance typhoid fever, or in the course of chronic diseases, like diabetes and chronic

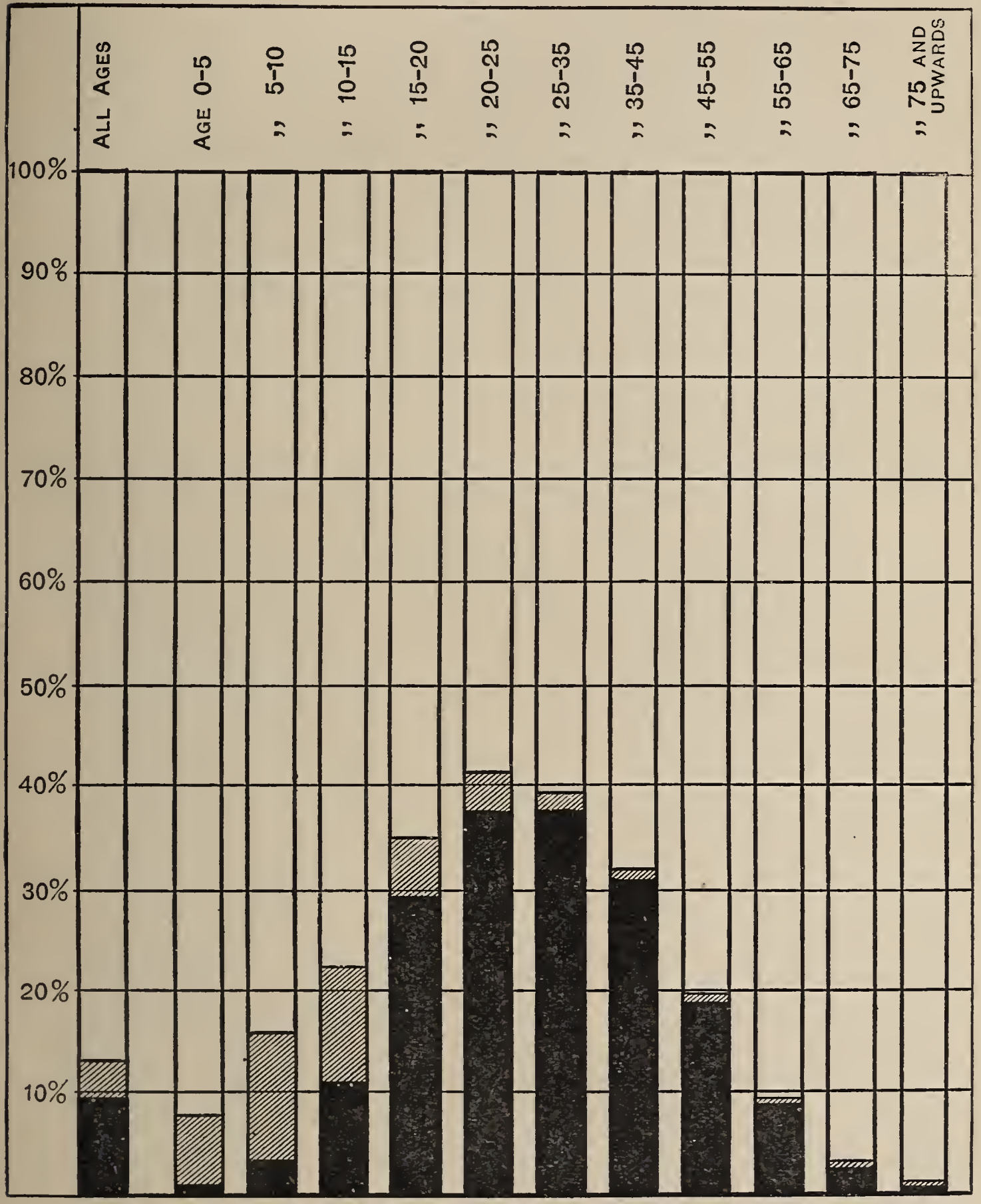


Fig. 125.

Diagram showing the proportions of deaths from tuberculous diseases per 100 deaths from "all causes" of each age-period (London, 1897–99). The black represents the deaths from phthisis, the shaded part the deaths from other tubercular diseases. (Shirley Murphy—Tuberc. Congr., London, 1901.)

alcoholism. To the same cause may be referred the phthisis met with in those who married too young, or who have had too large and rapid a family.

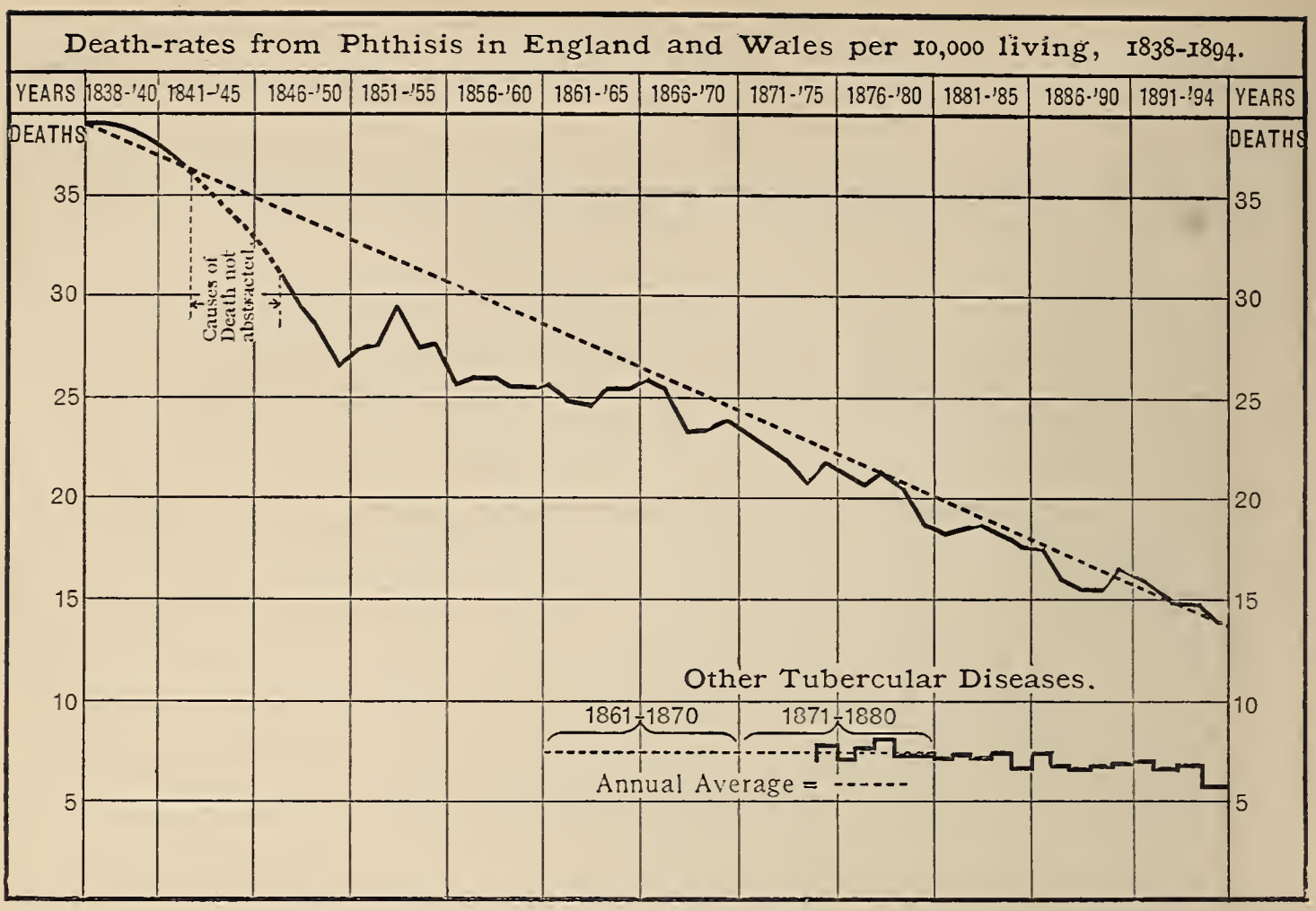


Fig. 126.—Death-rates from phthisis in England and Wales per 10,000 living, 1838–1894. (Ransome, *Lancet*, July 11, 1896.)

MORTALITY FROM PHTHISIS PER 1,000,000 LIVING, for the TWO SEXES

Contrasting the period 1851-60.... ——— Males — — — Females.
with the period 1896-99..... ——— Males — · — · — Females.

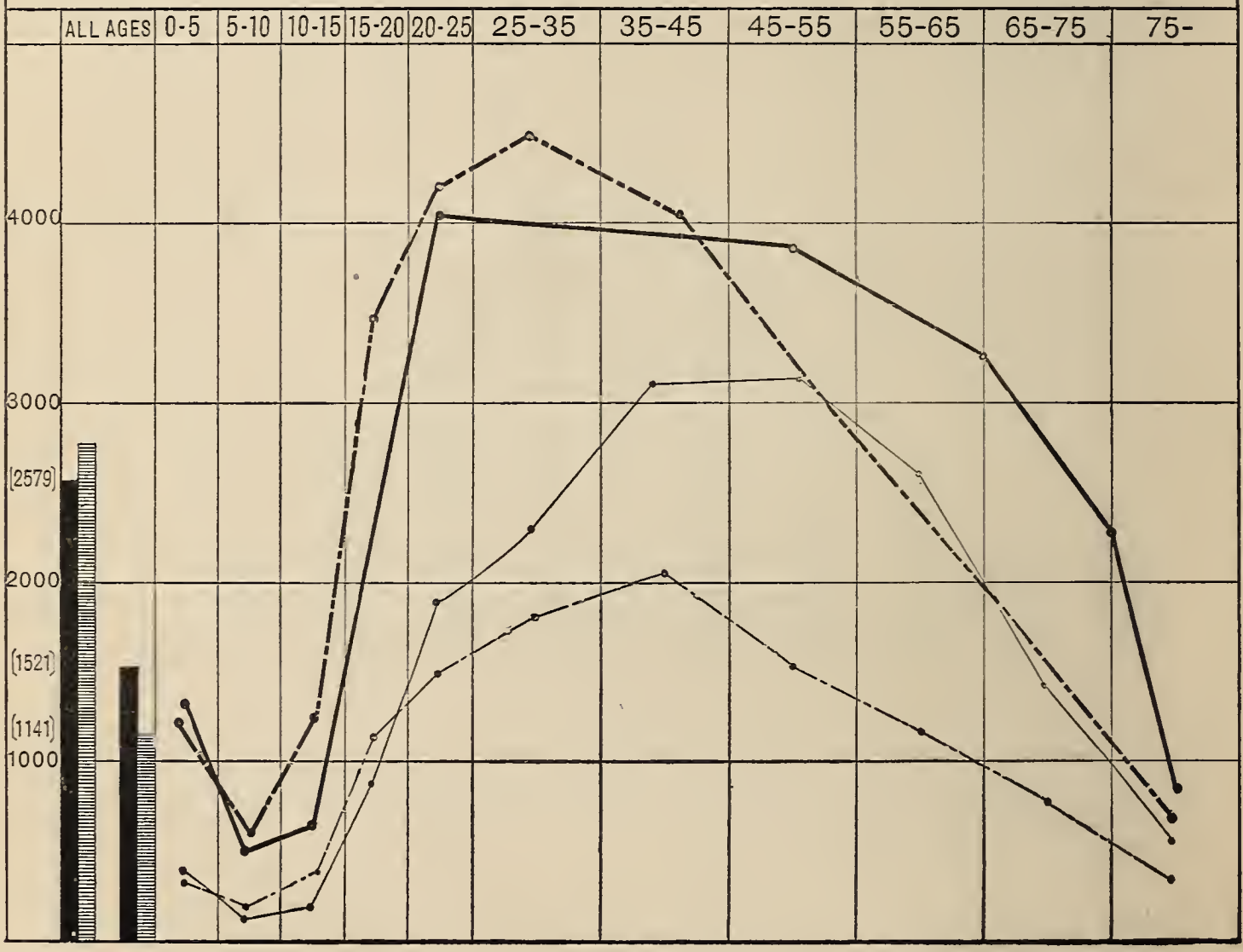


Fig. 127.—Diagram constructed from the figures given by Dr. John Tatham in his paper read before the Tuberc. Congr., London, 1901.

The diagram shows that a reduction has taken place in the mortality at each age-period, and that the reduction is greatest in females—the reduction for males being 41 per cent., and for females 59 per cent.—that is to say, for every 100 males who died of phthisis in the period 1851–60, only 59 die now, and for every 100 females who died in that period, only 49 die now.

It also shows that the period of maximum mortality from phthisis has been postponed in both sexes; for males, from 20–25, in 1851–60, to 45–55 now; for females, from 25–35, in 1851–60, to 35–45 now. This means either that the saving of life has been greatest at these periods, or that persons liable to phthisis have lived longer than they would under the earlier conditions.

Inheritance.—The recent additions to our knowledge of tuberculosis have greatly modified the views held as to the influence of inheritance in phthisis, for they have established the much greater frequency of acquired tuberculosis, especially in early life, and they have shown that we can only arrive at the true value of inheritance as a factor in phthisis by deducting from the cases of family phthisis those among them which are certainly or probably acquired.

Appeal is often made to large statistics, but frequently no account is taken of other possible determining causes, so that what is required to be proved by the figures is first assumed. Statistics establish no more than that there is such a thing as family predisposition, and show nothing as to what that predisposition consists in.

The effects of family predisposition upon phthisis may be estimated in two ways.

1. By taking a series of cases of phthisis at random and ascertaining the percentage in which a history of phthisis in other members of the family can be obtained.
2. By taking a series of tuberculous families and of non-tuberculous families and comparing the percentages of phthisis in the members of each.

The first line of investigation is that which has been most frequently followed. General statistics show that on the average about 28 per cent. of all cases of phthisis yield a history of phthisis in the parents, and about 25 per cent. more in collateral relatives more or less remote. It is obvious that the value to be attached to the two sets of figures is very different, for the chances rapidly increase, the wider the circle of relationship is made, that the phthisis has developed in the ordinary way and independently of family predisposition.

The percentages[1] of family phthisis, reckoning the direct and collateral lines together, are very variously stated as ranging from 30 to 87 per cent., but the higher statistics are of little value, for with a disease so common as phthisis there is hardly any family which will not show some instance of it among its more or less remote relatives.

For our present purpose it will be best to limit our consideration to family phthisis only in the direct line.

It is evident that, even if family predisposition be potent, it does not do away with the ordinary risks of phthisis to which every one is liable, and this we know to amount to something like 15 per cent. Deducting this from the 28 per cent. at which the rate for family phthisis was taken, we obtain 13 as the value of family predisposition as such.

This result is much the same as that at which Walshe arrived, for taking 25 per cent. as the average rate for parental inheritance and 15 as representing the general liability to phthisis, he thus obtained the figure 10 as the percentage value of inheritance.

[1] Tables given by Wilson Fox, p. 525.

If, then, 10 per cent., or, to be well outside the mark, 15 per cent., be accepted as the value of direct family predisposition, it follows that it does not do more than, at the outside, double the general liability to phthisis.

Reginald Thomson quotes from the records of some Insurance Offices to show that while the mortality from phthisis among those lives in which a family history of phthisis was given amounted to 15 per cent., it reached 10·8 per cent. in those in which there was no such family history. The difference between the two rates, viz., 5 per cent., represents the value of family predisposition in this class of case, but it must be remembered that it is a class of selected lives, from which all those with well marked-family history are excluded.

The experience of the New York Mutual Office, which consistently declines all lives in which the family history is in any way suspicious, shows that even so the mortality from phthisis reaches 17 per cent. The mortality in New York itself is 30 per cent., thus leaving again 13 per cent. as the value of family predisposition.

Although 10 or 15 per cent. may represent the value in general of family predisposition, it may run much higher in certain families, and in some cases amount to a veritable plague, cutting off one member after another in rapid succession.

Thus Reginald Thomson[1] gives a table of 80 families with 385 children of whom 50 per cent. were tuberculous. Austin Flint[2] refers to 13 families with 91 cases of phthisis among them, and of these 7 families yielded no less than 56 cases. Wahl[3] gives a record of 61 families with 184 cases of phthisis among them.

A few other facts about what is called inherited phthisis remain to be mentioned. Thus it is stated (1) that inheritance tells much more upon female than upon male children. Considering, however, that there is no special sexual predisposition to phthisis, this would imply that the difference was really due to other causes, *e.g.*, their mode of education and habits of life. Thus the boys are generally sent away early from home to school, and lead a hardier, more active life, while the girls are kept at home and lead a sedentary and confined life, and are, therefore, more exposed to the influence of local conditions. The statistics given differ widely, but the female excess is often stated as between 12 and 18 per cent.

(2) That it is more potent through the mother than through the father, and that fathers are more likely to transmit to sons, and mothers to daughters, than crosswise, but on these points, again, there is great divergence of opinion.

(3) That it hastens the period of attack by $2\frac{1}{2}$ years in males and $3\frac{1}{2}$ years in females (Walshe), or, according to Th. Williams, by 3 years and $6\frac{1}{2}$ years respectively.

(4) That it favours a special form of the disease ; but this is not peculiar, for a family type is seen in many other affections besides phthisis.

(5) That in phthisical families the children, even when they do not become phthisical, are liable to other forms of chest affection, *e.g.*, asthma, bronchitis, etc.

Family predisposition being thus an essential factor in phthisis, though probably not exerting so important an influence as has been hitherto believed, the question now arises as to what it is which is transmitted, whether it is the disease itself or merely the tendency or predisposition to it.

Inasmuch as phthisis may develop as the result of tubercular lesions in any part of the body—lesions which may have existed long antecedent to the changes in the lungs, and dating, it may be, even from childhood—the question of the inheritance of phthisis cannot be separated from that of tubercle.

Now we know that although tubercle is common in children, it is less frequent in infants than among children above one year of age, and that among infants it becomes more and more rare, the younger the infant is, *i.e.*, the shorter the time the child has lived.

Barthez and Rilliet[4] give the following table for tuberculosis in infants and young children:—

Up to 15 days old, ...	2	cases out of	385	=	0·5 per cent.
From 15 to 30 days, . . .	0	,, ,,	275	=	0 ,,
,, 4 months to 1 year, . .	8	,, ,,	141	=	5·5 ,,
,, 17 ,, 2 years, . .	8	,, ,,	82	=	10 ,,
,, 27 ,, 3 ,, . .	10	,, ,,	45	=	22 ,,

[1] *Family Phthisis*, p. 45.
[2] *Phthisis*, 1875.
[3] *D. med. Woch.*, 1875 ; *cf.* Wilson Fox, p. 529.
[4] *Dis. of Children*, p. 889.

These facts can be explained in one of two ways only; either the tubercle was acquired after birth; or, if present at the time of birth, it must require time to develop, *i.e.*, it must lie latent for some time.

How easily tubercle is acquired in the very young is proved beyond question both for man and animals, and it has been traced without doubt in most cases to the feeding with infected milk; above all, to suckling by a tubercular mother or nurse.

Further, there is evidence to show that if these sources of infection be eliminated, the influence of predisposition can be reduced to very small dimensions.

Bolitz's [1] statistics show the same thing.

Thus in Kiel, during the years 1873–1889, *post-mortem* examination was made of 2576 children, of whom 424 = 16·4 per cent., died of tuberculosis, which was distributed over the different ages in the following manner:—

Stillborn children,	0·0 per cent.	From 1–2 years old,	26·8 per cent.
Up to 4 weeks old,	0·0 ,,	,, 2–3 ,,	33·0 ,,
From 5–10 weeks,	0·9 ,,	,, 3–4 ,,	29·6 ,,
,, 3–5 months,	8·6 ,,	,, 4–5 ,,	31·8 ,,
,, 6–12 ,,	18·3 ,,	,, 5–10 ,,	34·3 ,,
		,, 10–15 ,,	30·1 ,,

Bollinger [2] records the experience of the Munich Orphan Asylum, in which, out of 613 children, over a space of twelve years, only one became tubercular, in spite of more than half of them having lost either father or mother, or both parents, from consumption.

We may now turn to the question as to what evidence there is to prove the existence of tubercle in the infant at or before birth.

Fœtal Tuberculosis.—In man fœtal tuberculosis is so rare that Virchow was led until quite recently to deny its existence; and but few conclusive cases have been recorded.

Heller [3] examined 300 stillborn children and found not one tubercular, though in 1 case the uterus itself was tubercular. In another series of 541 children who lived to the age of 9 weeks, only 1 was found tubercular.

Birsch-Hirschfeld [4] and Schmorl, in a seven months' fœtus, born of a phthisical mother, found tubercle bacilli in the placenta and liver, but the spleen and kidney, though showing no evidence of tubercle, were infected.

Bar and Renon [5] inoculated guinea-pigs with blood from the umbilical cord, and obtained positive results in 2 cases out of 5.

Though bacilli in the body of the fœtus are so rare, several cases are recorded in which both the fœtal and maternal parts of the placenta were tubercular.

Lehmann records the case of a woman, 41 years of age, who died, three days after parturition, of tubercular meningitis. The infant died 24 hours old, and tubercles were found in the liver, spleen, lungs, mesenteric and bronchial glands.

In cattle, the existence of fœtal tuberculosis must be acknowledged, but its rarity is also beyond question.

In Copenhagen [6] 16·28 per cent. of the cattle were tubercular, but only 0·12 per cent. of the calves (Bang).

In Kiel [7] 13 per cent. of all cattle slaughtered, but of the calves only $\frac{5}{6300}$ in one series = 0·079 per cent., and in another series of 8300, no case at all (Heller).

[1] *Journ. Comp. Path. and Ther.*, Dec. 1890, p. 370.
[2] *Münch. Med. Woch.*, 1888, Nos. 29 and 30.
[3] *Viertel-jahrschr. f. Gesundheitspfl.*, 1890, Heft i.
[4] Ziegler, *Beiträge*, ix. 428.
[5] *Ann. de Gynec. et d'Obstet.*, Sept. 1895. Johne for references. *Gesch. d. Tuberc.*, p. 77. *Berl. kl. Woch.*, July 9, 1894.
[6] *D. Ztsch. f. Thier. Med*, 1890, 355.
[7] *Loc. cit.*

In Berlin 4 cases in calves out of 15,400 (Johne).[1]
Köckel and Lungowitz.[2] In 2 tubercular cows killed during pregnancy, the calves of about the sixth month gave both macro- and micro-scopic evidence of tubercle.

Although the occurrence of fœtal tuberculosis must be accepted as proved, still its extreme rarity shows that this cannot be regarded as the ordinary mode of transmission.

It has, however, been suggested that the disease may be transmitted, and yet that the lesions may not develop until some time after birth, and in proof of this theory, Landouzy and Martin[3] quote their experiments to show that the tissues of fœtuses born of tubercular mothers are infective, though not yielding themselves any evidence of tubercular lesions.

As against these experiments must be placed the repeated failures to obtain tubercular fœtuses from parents rendered tubercular by inoculation, so that Landouzy's[4] experiments would require repetition and confirmation before they could be accepted as conclusive. The suggested analogy of syphilis is very misleading, and, at any rate, is useless as an argument upon a question of fact.

It has been suggested that the infection may be transmitted to the fœtus *in utero* from the father direct, without infection of the mother. This, again, is quite unproved, and against it stand numerous instances in which tuberculosis of the genito-urinary track has existed in the father without the fœtus having become tubercular, while, on the other hand, instances are not rare in which mothers with actual tubercular disease of the generative organs, *e.g.*, ovaries, tubes, or uterus have given birth to uninfected infants.

The direct transmission of tubercle itself from parent to child is, therefore, to say the least, not proved; indeed, the weight of evidence is strongly against it.

If the disease then be not transmitted, all that can be transmitted is the tendency to it, *i.e.*, in modern phraseology an increased vulnerability of tissues, or a diminished resistance to the bacillus.

The importance of this view in the management of a family with phthisical predisposition is too obvious to require further emphasis.

Constitution, Diathesis, Build.—Although tubercle itself is not inherited, still the delicacy which exists with family predisposition might possibly manifest itself in general build and constitution. For this the term strumous or tubercular diathesis was invented. It was generally described as seen in children or young persons. They were delicately and frailly built, of a clear and bluish complexion, with bright eyes and large pupils, long eyelashes and downy skin, and with thin and slender bones; they were of poor muscular power and easily fatigued; subject to catarrhs, both of the respiratory organs and bowels, and liable to glandular enlargements, both internal and external; and they often fell victims to phthisis.

So far as these lymphatic affections are concerned, we now know them to be tubercular, so that what used to be called strumous glands are proof, not that the patient is likely to become tubercular, but that he actually is so.

Attention has also been drawn since classical times to certain peculiarities in the chest and upper parts of the body in persons who become phthisical; viz., the roundness of the upper part of the thorax is lost, the upper ribs are flattened, the sternum depressed, and the intercostal spaces widened. This is the *flat chest*, the chief character of which is the reduction of the antero-posterior

[1] *Loc. cit.* [2] *Beitr. zur Path. Anat.*, xvi. pt. ii.
[3] *Soc. Med. d. Hop. de Paris*, April 1886. *Rev. de Med.*, 1888.
[4] *Cf.* Rep. to Internat. Veter. Congr., Brussels, Fleming, 1883.

diameter. The shoulders droop and fall forward, and often, in so doing, tilt the scapulæ out from the body, so that they project like rudimentary wings, a condition aptly described by the names *alar* or *pterygoid chest*. At the same time, the muscles about the neck and upper parts of the thorax are ill developed, so that the neck is thin and lanky. This is the condition indicated by the term *paralytic thorax*. All these forms of the chest were described by the common term *phthinoid*, *i.e.*, like those met with in phthisis.

The best instances of these forms of thorax are, of course, found in persons who are actually phthisical, but the term phthinoid was used to imply that the chest, though not the actual seat of phthisis at the time, was likely to become so in the future.

Whether this old belief be true, is difficult to prove, but it may be partly true on the general principle that ill-formed organs are especially liable to disease.

We certainly know that even in phthisical families the chest is often perfectly formed; that, where it is ill formed, many escape phthisis; and lastly, that phthisis is common enough in persons whose chests are perfect in form, and even remarkably well developed.

All that can be safely inferred, therefore, in respect both of constitution and conformation of thorax, is that frail and delicate persons with ill-formed chests are somewhat more liable than robust, well-developed persons to develop phthisis.

It is such a general frailty or delicacy which would explain the stated frequency of phthisis in the offspring of marriages of consanguinity, or of parents between whom there is great disparity of age.

Age.—In considering the relation which exists between age and phthisis, several questions arise, which, though really distinct, are frequently confounded. We have to consider the age-periods in relation with—

1. The prevalence of phthisis.
2. The mortality from phthisis.
3. The commencement of phthisis.

And in close connection with each stands the duration of phthisis, and the influence which the age or date of commencement of phthisis has upon it.

1. The *general prevalence of phthisis* is not easy to determine by figures, but there can be no doubt that phthisis is most common in early and middle adult life.

The analysis of 435 cases, taken as they presented themselves from my out-patients records at Victoria Park Hospital, shows the following age distribution :—

0–5	5 to 10		—15		—20		—25		—30		—35		—40		—45		—50		—55		—60		65		Above 65.
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	
0	2	3	2	5	20	22	47	43	53	40	43	25	45	7	18	15	17	8	11	2	4	2	1	0	0
0	5		7		42		90		93		68		52		33		25		13		6		1		0
							183				120				58										

Putting little children out of consideration for the present, phthisis is clearly uncommon before the age of 15. Between 15 and 20 it suddenly becomes frequent, and its prevalence reaches its maximum between the ages of 20 to 30.

After 30 its frequency declines each quinquennium, until after 50 it becomes about as rare as before 15. Phthisis is at its maximum between 20 and 40, during which period 66 per cent. of the cases are met with.

2. *The mortality of phthisis.*—The figures usually quoted are taken from mortality tables, but they show, as would be expected, the same thing, for it might be *a priori* assumed that the mortality would be greatest at those periods at which phthisis was most prevalent.

For the decennial period 1871–80, the Registrar-General's returns show that the percentage mortality of phthisis falls upon the different age-periods approximately in the following proportions :—

5–10	—15	—20	—25	—35	—45	—55	—65	—75	Above 75.
1·7	3·1	9·6	14·7	17·0	17·6	14·7	11·5	7·0	2·3
				49·3					

The returns show a gradually increasing relative mortality up to the age of 45, while out of 100 deaths from phthisis, 50 occur between the ages of 20 and 45. *Cf.* charts and diagrams under Etiology.

These facts are evident in all statistics, though the actual proportions stated differ a good deal according to the sources from which they are taken, the differences depending chiefly upon the relative frequency of phthisis in children.

Phthisis in Children.—In children, tubercular affections of the lung are frequent enough, but phthisis in the ordinary acceptation of the term, *i.e.*, in the subacute or chronic form so common in the adult, is without doubt comparatively rare. The discrepancy is to a great extent apparent, and is chiefly one of terms, affections, which in the adult would be placed under the heading of phthisis, being placed in the child by one author under phthisis, and by another under some other heading, for instance, under tuberculosis or broncho-pneumonia.

The differences between the tuberculosis of the child and of the adult are chiefly these.

Tubercle tends to be a much more acute and fatal malady in the child than in the adult.

It has a greater tendency to become disseminated, *i.e.*, general, and when the lungs become involved, they show the miliary granulation rather than the more chronic destructive lesions of phthisis. In the adult with tubercle in any part of the body, the lungs rarely escape. This, which is Louis' law, does not hold in children, for in them the lungs often escape, even to the extent of 28 per cent. (Barthez and Rilliet).

The chief difficulty lies in respect of broncho-pneumonia, and in determining how much of it is really tubercular. Some undoubtedly is, and probably more than has hitherto been believed, as recent observations based upon bacteriological investigation demonstrate (Landouzy).[1]

There can be no doubt that what in the adult would be called acute or galloping phthisis, is in the child often called broncho-pneumonia.

[1] *Bull. d. Soc. Med. d. Hop. d. Paris*, April 1886.

Closely connected with this is another question upon which there is much difference of opinion, and for the same reason, viz., the frequency of cavities in children, some authors stating that they are rare, and others making them common.

Barthez and Rilliet; 28·6 per cent.; West, 22·7.

All, however, agree that chronic cavities such as are met with in the adult are rare, and that the cavities found in the lungs of children are usually of small size, and for the most part only spots of softening in the midst of recent consolidation. Such cavities as these are not rare even in the very young.

Chronic cavities are recorded in a child of 9 months by Thomson[1] and in a child of 11 months by Norman Moore.[2]

Such chronic cavities may even lead to fatal hæmorrhage, as in a case recorded by Revilliod[3] in a child of 16 months, and by Cholmeley[4] in a child of 20 months; while in Fagge's[5] case an aneurysm of a branch of the pulmonary aneurysm was found ruptured, exactly as so often occurs in the chronic cavities of the adult.

3. *The commencement of phthisis.*—Phthisis may, of course, commence at any age, but there is general agreement that the most common period is that of early adult life, *i.e.*, between the ages of 15 and 30. After 30, the risk is much less, and becomes less and less as years advance. Still phthisis may develop quite late in life, even in the aged. Judging from my own experience, I should say that phthisis in elderly persons is liable to run an acute course, and to be rapidly fatal.

In this respect, like some other affections in the aged, phthisis seems to revert to the types met with in children, and to depart from that which prevails in the more vigorous period of life. As in children, so in the aged, it is stated that recent phthisis does not easily lead to the formation of cavities.[6]

It may, of course, be that the onset of symptoms of phthisis marks the date also of infection; but in most cases it is really either the recrudescence of old tubercular lung mischief, or infection of the lung from some old tubercular focus elsewhere.

The frequency with which the lesions of chronic or "healed" phthisis are found in the lungs of elderly persons has been already referred to. Attention has been drawn to the fact that acute tuberculosis of the lung is not altogether rare in the aged, though instances of phthisis actually commencing in the aged are rare.

Laennec records a case in a man at the age of 99; Durand-Fardel at 73 and 74; Andral at 66; and Pollock at the age of 60.

Other cases are mentioned in a note by Wilson Fox, but, in the reports given, the distinction is often not clearly drawn between old and recent phthisis.

Sex.—Dr. Ogle,[7] calculating from the death-rates, finds that the mortality over the whole of life in the two sexes is the same, but that at the different age-periods, the rates for the two sexes differ considerably; for while during the first five years of life they are practically identical, between 5 and 35 the rate for the female is much higher; thus between the ages of 10 and 20 it is nearly half as high again, *i.e.*, in the proportion of 3 to 2; after 35 the rate for the male is the higher, and remains so till the end of life.

[1] *Ed. Med. Journ.*, Oct. 1888.
[2] *Path. Soc. Trans.*, 1885.
[3] *Virch. Jahrb.*, 1887, ii. 713.
[4] *Path. Soc. Trans.*, xxxix.
[5] *Path. Soc. Trans.*, vol. xxviii.
[6] Moureton, *Thèse d. Paris*, quoted by Wilson Fox, p. 522.
[7] The paper is published *in extenso* in Wilson Fox, *Lungs*, p. 579.

The reason of this difference lies probably in the facts, that girls are brought up in a less healthy and hardy way than boys, that they are more exposed to the influences of unsanitary domestic conditions, and that while the strain of life falls most heavily upon women during the child-bearing period, it falls upon men at a later period.

In females, phthisis is said to begin earlier, and last a shorter time, *i.e.*, to run a more rapid course.

Inheritance has been said to tell more heavily on females than males, and reference has already been made when dealing with the question of contagion to the curious fact that as between husbands and wives, when the one becomes phthisical after the other, more wives fall victims than husbands.

THE GENERAL AND LOCAL SIGNS OF PHTHISIS.

Tubercle is essentially a local disease, but often associated with constitutional disturbance. The signs of tuberculosis of any organ fall naturally into two groups—(1) the local signs which indicate the seat of the disease and vary with the organs affected, and (2) the general signs or evidence of the constitutional disturbance. These are the same wherever the disease is seated, and may be the earliest manifestations of disease.

In dealing with phthisis, it will be most convenient to consider the general signs of constitutional disturbance first and afterwards the localising signs, *i.e.*, the symptoms and the physical signs.

A. THE GENERAL SIGNS.

Constitutional Signs.—Constitutional signs are rarely, if ever, absent throughout the whole course of the disease, for if not present at any given time, they either have been present or will develop.

They may be the first evidence of mischief, and may precede by some time the development of any localising signs. They do not stand in any constant relation with the amount of mischief in the lung, for they may be well marked when the physical signs are quite indefinite, and on the other hand may be absent when the physical signs show the existence of extensive disease. As a rule, however, they are well marked when the disease is progressing, and so they become of the highest importance in prognosis, for they may be taken as evidence that the disease is active, and in some degree serve also as a measure of that activity.

The constitutional signs of phthisis may be summed up in the words hectic fever, *i.e.*, they consist in irregular rises of temperature, increased rapidity of pulse and respiration, loss of flesh and strength, impairment of appetite and digestion, with irregularity of the bowels, associated in severe cases with sweating, shivering, vomiting and enlargement of the spleen.

In all these there is nothing peculiar to phthisis, for the same phenomena are seen in other fevers, notably those of a septic character, and in connection with suppuration.

In marked degree these symptoms occur only in the acute forms, and then phthisis may so closely resemble certain other fevers that diagnosis is often at first very difficult. Thus it comes that tuberculosis is confounded with pneumonia, typhoid fever, and internal suppuration.

Fever.—Pyrexia is a part of the tubercular process. Some, however, have gone so far as to maintain that a temperature of more than 100° or 101° is an indication of some additional septic infection; but this view can hardly be

maintained in the face of the fact that temperatures of 103° or 104° are not uncommon in acute tubercular processes elsewhere, *i.e.*, in the pleura or peritoneum, and that high fever is the rule in acute miliary tuberculosis of the lung.

Nor do I think that any special form of temperature-curve can be associated with the different forms of phthisis, as some authors maintain, at any rate in more than a perfectly general way; viz., that where the process is active or acute the pyrexia is high; where it is not very active, moderate; and where it is stationary, absent altogether.

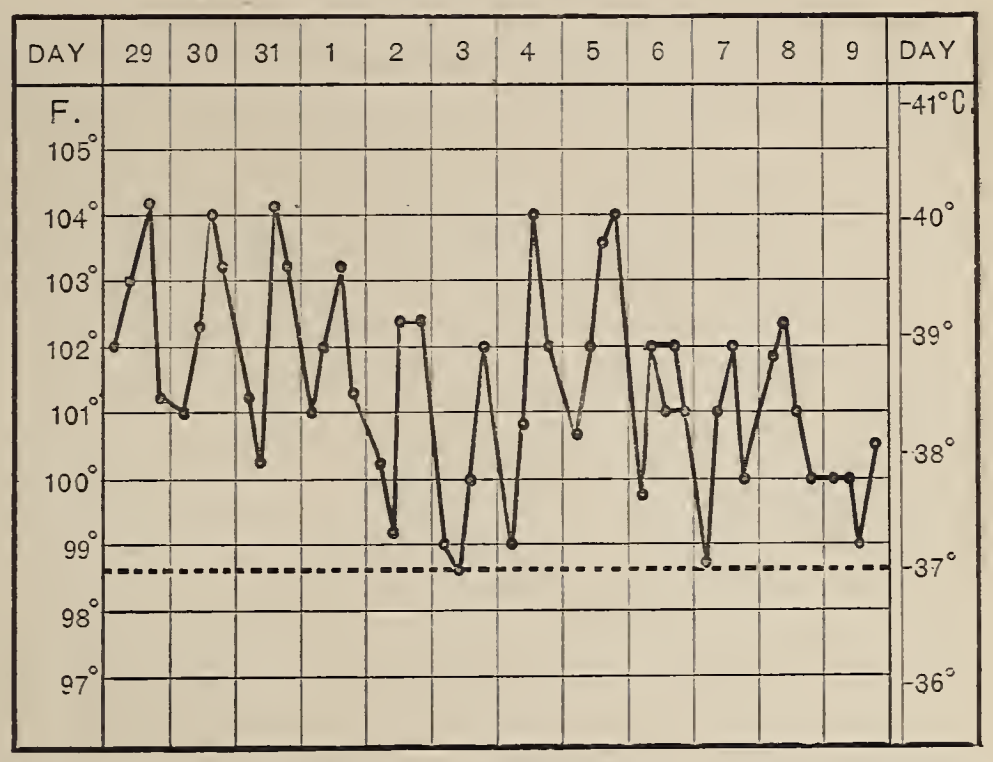


Fig. 128.—A. B., aged 20. Acute phthisis. Six-hour temperature chart. The case ran its course in about five months. The temperature continued much the same throughout.

The characteristic temperature-curve of phthisis is that of hectic fever, viz., long-continued pyrexia of great irregularity, and with wide daily oscillations.

The pyrexia is most pronounced in acute cases. It is absent where the disease is stationary; and may be present in any degree in the subacute and chronic cases, so that it may be taken as a general index of the activity of the disease.

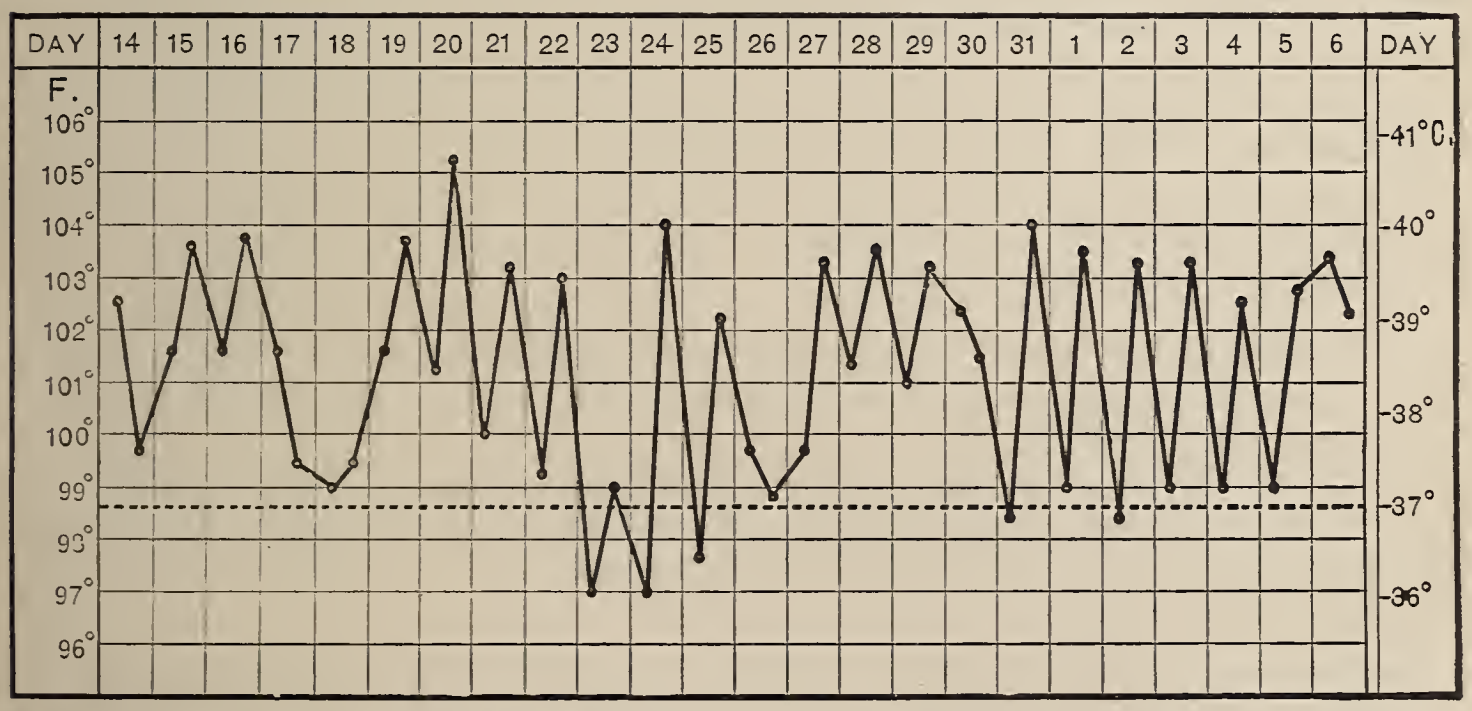


Fig. 129.—Charles G. Phthisis of twelve months' duration in an acute stage. The oscillations of temperature were considerable and continued until death.

It is usually highest in the after part of the day, and lowest in the early part, but the reverse (typus inversus) may be met with in phthisis, as in other fevers.

The actual time of the maximum varies, soon after mid-day, in the late afternoon or in the evening; but the common time is between six and eight o'clock in the evening.

The minimum is usually early, and often about two or three o'clock in the morning.

The pyrexia of phthisis is characterised by its great instability, that is to say, it rarely remains long at the same level, but is always either rising or falling, and sometimes shows several minor daily oscillations, besides the great ones so obvious on the ordinary eight hours' chart.

The maximum is not, as a rule, above 103° or 104°, unless in very acute cases, or in connection with complications. Temperatures of 106° or 108° are sometimes met with, but as a rule only towards the end, and shortly before death.

The minimum is often much below normal, so that the daily oscillations, *i.e.*, the difference between maximum and minimum, may be considerable. A difference of 6 or 8 degrees is not rare, and it may be as much as 10 and 14 degrees.[1]

It has been stated that the temperature may differ on the two sides of the body, being higher on the side where the disease is most active. This is purely an accident and of no significance, and it is seen in other diseases than phthisis, and may occur even in healthy persons.

The pyrexia varies to some extent with idiosyncrasy, *i.e.*, some persons fever more easily than others. They are usually nervous, excitable persons, in whom the temperature runs up to unusual heights on slight provocation. When they are attacked by phthisis they offer but little resistance to the disease, which, as a rule, runs in them a rapid course.

Complications may send the temperature up or cause it to fall. Thus, with pneumonia, pleurisy, or other acute inflammation, the temperature will rise and run the course usual in those affections. On the other hand, such complications as pneumothorax, or profuse hæmoptysis, from the collapse or shock they produce, may cause the temperature to fall considerably.

Other things being equal, it is the mean or average temperature which gives the best indication of the course the disease is taking. Thus a continuously high mean or a rising grade indicates active progress, and a low mean or falling grade, retrogression. In the later stages the instability of temperature becomes more marked and the oscillations greater, until as death approaches the temperature may either rise to extreme heights, *e.g.*, 106° or 108°, or, as is more common, fall much below normal.

The sensations felt by the patient are little guide as to the amount of pyrexia, for as often as not they feel better rather than worse when the temperature is high.

Chilliness or *shivering* may be sometimes complained of, at or before the rise in temperature, but more often after an attack of sweating. Actual rigors are very rare, and usually due to complications, *e.g.*, to suppuration or pent-up pus in the pleura or in the lung.

Apyrexial phthisis has been described, *i.e.*, progressive phthisis without rise of temperature. It must be very rare. Large cavities in the lung are, of course, often met with a normal temperature; but in such cases the mischief is of ancient date, and the disease at the time is quiescent; if it become active again the fever returns. This has been called *latent phthisis*.

[1] *Cf.* W. F., note to p. 808.

The following is the only instance of anything like apyrexial phthisis which I have seen.

Eugene S., aged 24, in whom phthisis had developed about 18 months before he was seen by me, had advanced phthisis of the whole of the right lung and of the upper lobe of the left. He was under observation for five weeks in the hospital, and though the disease was making rapid progress the temperature was subnormal throughout, except on four evenings, when it reached 99°, and on one when it reached 100°.

It sometimes happens that the temperature, though raised at some time during the day, remains above normal so short a time that the rise is easily missed.

I remember one case in which I felt convinced there was fever, but I could get no evidence of it until I tied a thermometer into the axilla, and left it there for twenty-four hours. I then found that it had risen to 103°, but only for so short a time that it had been regularly missed, though the temperature had been taken, in the ordinary way, three or four times a day.

Loss of Flesh.—Loss of flesh is almost an essential part of phthisis. It may be the earliest obvious sign of mischief, and may precede the fever. It may, at any rate, be well marked, when the temperature is low or only occasionally raised.

Loss of flesh forms a good measure of the activity of the disease; for it is rapid in the acute cases; it may be arrested if the disease become less active; and in very chronic or stationary cases the nutrition may be regained, so that the patients may be even plump or fat. However, the flesh, once lost in phthisis, is rarely completely regained, even if the disease become quiescent, but the patients remain thin and shrivelled. In the end the emaciation is extreme, until the patient is reduced to little more than skin and bone.

In the emaciation of phthisis many causes play their part; loss of appetite, impaired digestion, diarrhœa, profuse sweating, loss of blood and possibly affections of the lymphatic apparatus. Perhaps, also, the defective oxygenation consequent on the impaired respiratory processes takes its share; but all these are occasional and accidental causes only, for the emaciation is often greater than any of them can explain. There can be no doubt that the emaciation is connected with the disease itself, and it is probably the result of some poison produced by the tubercle bacillus.

The loss of weight is often very rapid.

Pollock[1] gives cases of a loss of 4 stones, and $3\frac{1}{2}$ stones, in 3 months, of 21 lbs. in 5 weeks, etc.

The net loss may in the end be very great, even as much as $\frac{1}{4}$ to $\frac{1}{3}$ of the total weight of the body in health.

The case of Freeman,[2] the prize-fighter, who died from phthisis after three years' illness, having lost $4\frac{1}{2}$ inches in height and nearly 48 per cent. in weight, is often quoted.

Under favourable conditions, even when the disease is progressing, flesh may be gained, as, for instance, when the patient is kept in bed and liberally fed.

Thus I have seen[3] a patient gain 9 lbs. in five weeks, and that while the mischief in the lungs was making active progress.

Instances of this are naturally more likely to be met with among the class of hospital patients than among the well-to-do.

Gain of flesh is always, so far as it goes, a good sign.

[1] *Progn. in Consumption*, 1865, p. 140.
[2] Hutchinson, *Cycl. Anat. and Physiol.*, iv. 1078.
[3] *Cf.* Wilson Fox, p. 833 note. For other instances, Williams, *Med. Chir. Trans.*, lviii

Loss of Strength.—Loss of strength and vigour may be the first indications of failing health of which the patient becomes conscious. The work which was once easy is now felt to be arduous. The sense of weariness and fatigue is ever present, and even sleep does not bring the customary refreshment, so that even after a long night's rest the patients rise unwillingly and unrefreshed. They look, as they say they feel, weary, languid and ailing. The sense of failing power often produces a condition of restless irritability, which, strange to say, usually passes off as the disease becomes more pronounced, and is replaced by the placidity and cheerfulness so characteristic of the disease.

The failure of health is often more obvious to those about them than to the patients themselves, who often refuse to acknowledge it, and from whom the history of it can only be obtained by cross-examination and close enquiry.

Night- or Sleep-Sweats.—Sweating is a very frequent though a very variable symptom in phthisis. It may occur early in the disease, even before definite physical signs are discovered; it may be present and well marked for a time, and then without any obvious reason disappear, to return again later. As a rule its disappearance coincides with general improvement, but not invariably. Though phthisical patients, like other feeble persons, may sweat easily on exertion, still the characteristic sweats occur at night, or, rather, during sleep, so that they might be more appropriately called sleep- rather than night-sweats.

Sometimes slight and but little complained of, they may be profuse, *colliquative*, as they have been termed. They then produce great exhaustion and discomfort, and are a source of danger by increasing the risks of chill. They may be so profuse as to soak the night clothes or even the sheets through, and to necessitate a complete change, it may be, even more than once in the night. Such profuse sweating is, however, not as a rule continuous during sleep, but usually, after the first copious sweat, the patients sleep the rest of the night without any repetition of it.

Sweating is usually most marked in the acute disease, and is therefore associated with the other signs of active progress, notably with fever.

Its relation to the temperature is not constant. It is more likely to be present the more hectic the type of fever is, but it comes and goes in the most irregular way. It may be present when the temperature is low, and absent throughout even when the temperature is high. It is most often absent in the acute cases, which present more the characters of acute broncho-pneumonia, and as this is the form which phthisis commonly assumes in children, it may be for this reason that sweating is less frequent in them than in adults.

It may alternate with profuse discharges from other parts, and thus cease for the time during profuse diarrhœa, and be absent when phthisis occurs in association with diabetes insipidus. On the other hand, in diabetes mellitus the appearance of sweating may be the first sign of the development of phthisis. The sweating affects chiefly the head and chest, but when profuse, involves the entire body. Hemidrosis has been described, but its association with phthisis is purely accidental.

Sleep-sweats are not peculiar to phthisis, but may occur in almost any exhausting disease, and are not uncommon in simple debility without any organic disease at all.

Thus I have frequently seen it in men worn out by overwork, and in women exhausted by hyperlactation, the symptom entirely disappearing, with rest in the one case, and with weaning in the other.

The cause of the sweating is still to be discovered. It has been referred to the fever and compared with the sweating of ague, but its relation to the fever

is inconstant, and it quite as often precedes a rise of temperature as follows it. It is most probably a toxic symptom, and due to stimulation of the secretory nerves or sweat glands by some poison circulating in the blood. The sweat glands were stated by Buhl to undergo fatty degeneration, but this has not been confirmed. In spite of the profuse sweating, sudamina are rare, which is interesting in contrast with their frequency after the sweating of rheumatic fever.

Shivering and Frontal Headache are signs of sudden constitutional disturbance, and are, therefore, rarely met with except in those cases of phthisis in which the onset is sudden, like that of pneumonia.

Headache is generally connected with digestive disturbance unless it depend upon meningitis, and it usually yields readily to simple treatment.

Rigors are very rare unless they mark the onset of some complication; slight shiverings or feelings of chilliness, however, are not uncommon, but they stand in no relation to the fever and are generally the result of sweating.

B. THE LOCALISING SIGNS.

The localising signs, *i.e.*, the signs which show that the respiratory organs are the seat of the disease, fall into two groups:

1. The symptoms, of which the patients complain.
2. The physical signs.

1. **SYMPTOMS.**—The symptoms are cough, shortness of breath, expectoration, hæmoptysis, and pain.

Cough is an almost constant symptom, and may precede by some time all other signs of disease. As a rule it is loose and easy, and does not give much trouble to the patient, but it varies greatly in different cases, and in the same case in different stages of the disease.

At first it is short, hacking, and may be paroxysmal; there may be no expectoration, or if any, it is scanty, viscid and difficult to get rid of. A little later the secretion becomes more abundant, and the cough looser and easier. In the later stages, when the sputum is more abundant, the cough is more frequent. Other things being equal, the cough varies in frequency and character with the amount and character of the expectoration. But it also varies with the part of the respiratory organs which is involved. Thus in the early stage, when the lesion is confined to the lung tissue, and the bronchi are little if at all affected, the cough is dry, hacking, short, but often painful by reason of the pleura being involved.

If the bronchi be involved, the cough has the character of that in bronchitis. It is more frequent, prolonged and paroxysmal, and often worse during the night and in the early morning. When the larynx or the bifurcation of the trachea is affected, it is often violently paroxysmal and very frequent, and in the former case it is, like the voice, hoarse.

When large cavities have formed and secrete freely, cough may be absent for hours while the secretion is accumulating in the cavity. As soon as the cavity is filled, or when, from change of position or other causes, the secretion overflows into the tubes, a paroxysm of coughing comes on, which does not cease until all the secretion has been discharged, and then peace may ensue until the cavity again becomes full and discharges itself.

As a rule, the cough, though frequent, does not give much trouble; but it may be a very serious complication by disturbing the patient's rest, and from the pain or exhaustion it causes.

Pain on coughing is usually felt over the seat of lesion, and is due to pleurisy. It may be muscular, and is then felt over the strained muscle wherever it may be, but the common seat is over the lower ribs or along the costal arch, where the abdominal muscles are attached.

Paroxysmal cough, when not due to the periodical discharge of secretion from a cavity, is most frequent where the trachea or bifurcation of the trachea is the seat of ulceration; but it has also been referred to the irritation set up by enlarged bronchial or mediastinal glands.

The cough is increased

1. By any cause which increases the respiratory movements, such as exertion, speaking, excitement;
2. By any respiratory complication, *e.g.*, bronchitis or laryngitis;
3. By change in position, especially where there are large cavities in the lung; and
4. Lastly, by food.

The taking of food may excite cough in several ways—(1) by irritation of the parts above the glottis, where they are the seat of inflammation or ulceration; (2) by the movements of the œsophagus or pharynx, where the larynx or trachea is ulcerated; (3) by direct pressure upon the trachea or bronchi by enlarged glands as the lumps of food pass by them; (4) by the temperature of the food; and (5) by the distension of the stomach, which may act either reflexly through the vagus or by direct pressure upwards on the diaphragm.

Violent coughing in phthisis, like any other paroxysmal cough, may lead to vomiting, just as it may to involuntary passage of urine or fæces.

Lastly, there are certain rare cases in which the disease may run its course to the end without any cough at all (*cf.* cases without sputum).[1]

Sputum.—The sputum varies a good deal in amount in different stages of the disease; this depends chiefly upon the amount of bronchitis with which the disease is associated.

In the very early stages there may be none at all, or at the most a little viscid mucus of quite indefinite character. As soon as breaking down has occurred, the characteristic sputum appears, and continues to increase as the disease progresses. When cavities have formed, the sputum becomes nummular, or, if the cavities be large, it may become purulent, almost like the discharge from an empyema, and may be brought up in gushes in considerable quantity at a time.

There are cases in which, from beginning to end, there is no sputum at all. In many of these the secretion from the lungs is scanty, and what little there is the patients swallow and will not, or do not, expectorate. But besides these, there are a few rare instances in which there really is no secretion at all.

A young man, with rapidly advancing phthisis, under constant observation in the hospital, coughed hardly at all, and brought up nothing. He was willing to expectorate it, but there was nothing. It was a very acute case of a few weeks' duration only, and when he died his lungs were found in a condition of acute caseous pneumonia.

I made also a *post-mortem* on a similar case at Victoria Park Hospital, and in that case the whole of one lung and half of the other was simply solid with caseating pneumonia, and the only cavities that existed were two or three minute ones, not larger than a haricot bean, in one apex.[2]

I also attended a young lady of 16 years of age, who died of acute phthisis after an illness of only three months' duration, who had neither cough nor expectoration until a week before her death.

[1] *Cf.* Lebert, *Brustkrankh.*, p. 181.

[2] *Cf.* Walshe, p. 17.

In other respects the characters of the sputum will vary with the complications that occur, *e.g.*, bronchitis, gangrene, pneumonia, or hæmoptysis. Under peculiar circumstances it may contain unusual adventitious substances, such as casts or calcareous masses.

The characteristic sputum of phthisis has a yellowish-green colour, and a faint sickly odour. It is viscid, coherent, and contains little or no air. It is usually scanty. It is expectorated in small lumps, and is described as nummular.

Nummular sputum is not pathognomonic of phthisis, but may occur in a discharging empyema, with cavities of other than tubercular origin, and with some forms of bronchitis, especially of the large tubes.

The amount is usually small, on the average 1 ounce, or 2, in the twenty-four hours, and there may be no more than a few pellets expectorated daily. It rarely exceeds 3–4 ounces. When much bronchitis is present, the quantity will be much increased, and in the midst of the more abundant bronchitic sputum, the phthisical sputum will appear in the characteristic streaks or lumps.

If there be large cavities which freely secrete, the sputum becomes more abundant, more fluid, and more purulent, resembling closely that met with when an empyema discharges itself through the lung.

If one of the characteristic pellets be examined, it is found to be composed of a basis of mucus, in which are entangled cells, caseous substance, granular detritus, crystals, fragments of lung-tissue, and bacilli.

The *cells* are chiefly small round cells, leucocytes or pus cells, with a few large epithelial cells, some columnar and occasionally ciliated cells from the bronchi, a few squamous cells from the mouth, and at times a few red blood cells even when the sputum is not blood-stained. Lastly, the shrivelled, granular, dark-coloured cells known as the tubercle-corpuscles of Lebert. Some of the larger cells are multinucleated, and these, as well as some of the smaller ones, contain pigment. The so-called myelin cells, *i.e.*, cells which have undergone myelin degeneration, are probably all derived from the goblet cells of the bronchi; they are not peculiar to phthisis, but are present in almost every kind of sputum, especially in that of bronchitis.

Caseous substance is found in more or less amount in different cases, sometimes in masses easily recognised as such by the naked eye, more commonly in minute lumps or streaks. The granular detritus is mainly formed of disintegrated caseous substance.

Crystals are sometimes found where the sputum has undergone degeneration, *i.e.*, triple phosphates, oxalates, tyrosin, fatty acids, and occasionally cholesterin and leucin.

Elastic fibres or *shreds of lung-tissue* are not rare, especially in rapidly destructive cases, but they are not always easy to find. For this purpose it is best to warm the sputum for a short time with caustic potash, and after allowing it to stand, to examine the sediment. Elastic fibres are characteristic enough with their sharply defined margins and curly ends. They are almost pathognomonic of phthisis, although occasionally met with in other destructive diseases of the lung, *e.g.*, gangrene and abscess. They may also be derived, it has been stated, from laryngeal ulceration.

Tubercle bacilli are pathognomonic and are very rarely absent. They are often distributed throughout the whole sputum and are easy to find; but they may be far from numerous and difficult to discover, and are then easily overlooked by not being searched for in the right part. They are undoubtedly sometimes absent, but this is distinctly rare, and, if not found on one day, they will be found sooner or later if careful examination be made. Being chiefly found in the caseous lining of cavities, they will be most abundant where this substance is freely expectorated, and are best looked for in the yellow lumps, spots, or streaks in the sputum which are composed chiefly of caseous substance. In old tubercular cavities which are stationary, or in the secretion which comes from them, there may be no bacilli; and again, in the very acute inflammatory cases, theoretically they might be absent, for there might be no breaking down, but, as a matter of practice, they are usually abundant. They may occur singly, or in twos or threes, or in groups, and now and then in masses large enough to be visible, when stained, to the naked eye.

Speaking generally, the more abundant the bacilli are, the more active is the disease, but there are many exceptions to this rule, and, as mentioned, some of the most acute cases have no sputum at all.

Besides tubercle bacilli, the sputum contains many other organisms, some possibly derived from the lung and air passages, but most of them from the pharynx and mouth.[1]

The *green colour* was referred by Trousseau to the action of organisms, but it may be due also to altered blood-colouring matter.

The *chemical examination* yields nothing characteristic; albumen, mucus, and the products of degeneration, fat, cholesterin, tyrosin, etc.

In diabetic phthisis sugar has been described in the sputum.

Extraneous substances may gain access to the sputum. These are chiefly derived from the food, such as milk globules, fat, shreds of meat, fragments of fruits, stains of beef-tea, tea, coffee or wine, giving a colour as if of blood, starch granules, fibres of linen or cotton. Most of these things are easily recognised if the possibility of contamination of the sputum with them is not forgotten. Thus I have been shown a small green common garden slug which the patient was said to have coughed up from the lungs, and also a piece of grape skin which was thought to be a blood clot.

Fœtor is rare in phthisis, and is due, no doubt, to a special infection. It may be associated with a slough or with gangrene of the lung, or it may be due to the decomposition of the contents of a cavity, or to the complication of a fœtid empyema. Once present it usually persists, but it may be transient, or come and go. In such cases the sputum may be otherwise unchanged in character, but if gangrene be the cause, the sputum becomes more fluid, of a dirty green colour, and contains the foul fœtid lumps characteristic of the condition.

Bronchial casts.—These are of two kinds, viz., fibrinous and blood, both rare.

Fibrinous casts are very rare in phthisis. A closer relation between phthisis and plastic bronchitis has been assumed than really exists, probably because of the frequency with which hæmoptysis occurs at the time the bronchial cast is expectorated. But hæmoptysis may occur in plastic bronchitis, and even be profuse, in patients who are not at the time, and never become, phthisical, and on the other hand, when plastic bronchitis is associated with phthisis, there may be no hæmoptysis.

The statistics[2] I have already quoted show that in 83 cases of plastic bronchitis, phthisis was actually demonstrable, or might be fairly assumed to exist, in 14 instances, *i.e.*, in 16 per cent. In Wilson Fox's series of 51 cases, phthisis was found in 7, or 14 per cent. Of these 4 were fatal, in 3 of which recent tuberculosis was found, and in the fourth chronic phthisis.

Blood Casts.—Blood casts in phthisis are very rare. Indeed Peacock[3] denied their existence entirely. They certainly occur, and I have myself seen two or three cases. A most remarkable instance was exhibited by me before the Pathological Society in 1880.

A man, aged 43, under treatment for phthisis, had two or three attacks of hæmoptysis, in one of which he brought up some "lumps" which he had expectorated with very great difficulty but with great relief to the dyspnœa, with which the hæmoptysis had been associated.

Four of the casts were shown to the Society. Three were of about the same size, viz., about 2 inches long and a quarter of an inch in diameter at the thickest part of the stem. Two of them were much branched. The fourth cast was much larger, being of the size and shape of the little finger, 1½ inches long and half an inch in diameter; this was not branched. The patient was under observation for twelve months subsequently. He spat no more "lumps." He died at home about three years later.

Fagge states that there is a specimen in Guy's Hospital Museum of a large branched clot expectorated a few days after hæmoptysis. A case is also given in abstract in Virchow's *Jahrbuch*, 1885, ii. 152, occurring in a phthisical diabetic who had had several attacks of hæmoptysis.

Cheyne,[4] who described blood casts in 1809, stated that they were the precursor of fatal hæmoptysis, and he quotes a case in support of his statement.

Small casts I have seen on two other occasions in phthisis. The most interesting of these two cases occurred in a man of 46, who had emphysema and also slight consolidation at the apex. He had several attacks of hæmoptysis while under observation, and he brought up small clots (bronchial casts) on two occasions; on the first he brought up about thirty small ones in the course of two days, on the second only two or three; the blood was at first bright and when the clots came it was dark.

Pomies[5] also records a case in which a large clot was brought up from the large bronchi of the left side, and quotes also another case in the practice of Gintrae.

[1] Baumgarten. [2] *Cf.* Art. on Plastic Bronchitis. [3] *Path. Soc. Trans.*, vol. v.
[4] *Path. of the Membrane of the Larynx and Bronchi*, 1809. [5] *Lyon. médic.*, 1872, x. 99.

Calcareous masses are occasionally brought up from the lungs.

They are usually of a grayish white colour, gritty and hard, but sometimes of a soft putty-like consistency. They are composed of phosphate, and sometimes, it is said, of carbonate of lime.

They vary greatly in size and shape. They may be minute and not larger than a caraway seed, or as big as a nut and weigh many grains. They may be cylindrical, oval, globular, pyriform, or quite irregular in shape, and they may also be branched. Their surface may be smooth, but it is usually rough and ragged.

The lung lesions with which they are associated, if any, are those of chronic phthisis, but calcareous masses may be expectorated for years and yet no signs of pulmonary disease of any kind become manifest.

They vary also in number from a single one up to many hundreds, *e.g.*, 500 in a case recorded by Portal.

The frequency with which such expectoration occurs is small. I only know of one set of figures which deals with this question, and that shows a frequency of 16 in 1000 cases of phthisis, but I think this yields an unusually high percentage.

Source.—Early writers[1] supposed that these masses were calcified or cretaceous tubercles, and thus Bayle and Portal speak of "phthisie calculeuse." Andral, whose description of the affection still remains one of the best, referred these masses to four sources.

1. The interior of cavities in the lung.
2. The lung tissue itself.
3. Calcified bronchial cartilages.
4. The interior of bronchial tubes.

It is strange that he should omit from the list the mention of calcareous bronchial glands, from which source most of the masses undoubtedly come ; especially as in one of the earliest cases recorded by Bayle *post-mortem* examination showed the presence of calcified bronchial glands.

There is, I believe, no pathological evidence to show that the contents of the bronchial tubes, whether ordinary bronchial secretion or blood, can inspissate and calcify. Even where the mass is branched, the branching is somewhat fanciful and not other than might be obtained from an irregularly calcified gland, for in such glands the deposit of lime salts is often very irregular.

Calcified bronchial cartilages we should *a priori* expect to be but rarely expectorated. Case 8 is an instance of this kind.

Calcified tubercular nodules might of course be expectorated when the lung tissue round them has broken down, but this explanation cannot fit most of the recorded cases, in which the signs of phthisis were either absent entirely, or, if present, showed the disease to be in the chronic and not in an active stage. Against this origin is the fact that most of the calcified tubercular nodules found in the lung are really formed of fibrous tissue in intimate connection with the rest of the lung tissue, and but rarely surrounded with a capsule from which they could be shelled out.

Although the disintegration of old cretaceous masses may be held to account for some of the cases of expectoration of calcareous masses, still the majority are to be referred, I think there can be little doubt, to the bronchial glands.

Calcareous glands are sometimes expectorated bodily, and the only difference between this group of cases and those under consideration is that the disintegration is fragmentary and the discharge piecemeal. For this view there is the most pathological support. Thus Fagge records a case in which a man had expectorated calcareous masses for some time at the rate of two or three a week, and after death a calcified gland was found lying in an abscess communicating with a bronchus. In all cases alike, whether the source be in the glands or elsewhere, the detachment must be brought about by a process of ulceration, and thus the expectoration of such masses will always be attended with risk, and it is often accompanied with more or less hæmorrhage. When it occurs in phthisis it is another evidence of the activity of the disease, but in many instances it is unattended with symptoms or physical signs, and the patients may remain in good health and otherwise unaffected for many years.

The following is a list of some of the cases I happen to have found recorded :—

1. Bayle.[2] Male, 59. *P.M.* calcified bronchial gland.
2. Portal. A case in which 500 were brought up over a long period of time.
3. Hamilton.[3] Female, 22, with much hæmoptysis and cough, the masses being mostly of small size, about that of a caraway seed. The largest was an inch long and irregularly branched, and a drawing of it given.

[1] Williams, *Pulmon. Consumption.*
[2] *Rech. s. la phth. pulmon.*, p. 286.
[3] *Dubl. Hosp. Gaz.*, 1854, i. 138.

4. Hamilton. Male, one mass twelve years before, patient still well.
5. Hamilton. Female, 17. Several with hæmoptysis. Pulmonary symptoms, but no signs of phthisis.
6. Guiburt.[1] Male, 34. Many attacks of inflammation of the lungs from the age of 6 years. One mass weighing 4·7 grammes. Patient died at the age of 34. No *p.m.*
7. Fredault.[2] Male. Calcareous mass found in bronchus at junction of middle and lower lobes, weighing 139 grains.
8. Henocque and Leroy.[3] Male, 21. Twelve calculi found in bronchus of right lower lobe, thought to be cartilages, and cartilage and bone demonstrated on section. Drawings given.
9. Peacock.[4] Male. At 22 slight hæmoptysis. At 23 chalky concretion expectorated, weighing 30 grains, preceded by pain in the left hand and arm. At 26 and again at 28 similar attacks. A few yellow masses were also expectorated which were thought to be caseous substance. No physical signs developed, and the patient was known to be well at the age of 30.
10. Walshe refers to cases, but describes none.
11. Burdel.[5] Female, 57. Irregular cylindrical calcified mass the size of a bird's egg, thought to be a calcified blood clot dating from hæmoptysis, twenty-two years previously.
12. Flint.[6] Male, 42 ; a great many masses ; patient in good health many years after.
13. Flint. Male, 23. A few small ones. Phthisis at the left apex.
14. Fagge.[7] Case already referrred to.
15. West. Case already referred to.

Hæmoptysis, though very common in phthisis, is not, as a rule, of frequent occurrence in any given case. It may be absent throughout, but if it has occurred once it is likely to occur again at some time or other, and it may be so frequent as to constitute the prominent symptom of the case, and this peculiar liability to hæmoptysis sometimes runs in families as other peculiarities do.

John H., aged 51, with bronchitis and emphysema, but no evidence of phthisis, was under my care for profuse hæmoptysis, of which I saw two attacks. He stated that he had had hæmoptysis twenty or thirty times, the first attack nineteen years before I saw him. He had brought up a pint or more on several occasions. His mother brought up blood in the same way for many years, and also two of his nieces (his sister's children). He himself had also lost blood occasionally from the nose and rectum, but there did not appear to be any other evidence of hæmophilia in himself or his relatives.

Hæmoptysis is stated to be absent throughout in from 20 to 30 of all cases of phthisis.

Statistics as to the frequency of hæmoptysis or of its absence are necessarily somewhat unreliable, and differ greatly according to the experience of different writers, on account of the long duration of the disease, and of the difficulty of watching the cases through the whole period of their illness.

The combined statistics, quoted by Wilson Fox, yield a percentage of 72 for its occurrence, leaving, therefore, 28 as the percentage of its absence.

Out-patient practice enables us to form a better estimate of the probabilities of obtaining a history of hæmoptysis in any given case as it presents itself in practice. Thus out of 420 cases of phthisis taken, as they came to me at Victoria Park Hospital, I failed to obtain any history of hæmoptysis in one-third.

A classification of hæmoptysis according to its amount may be usefully made.

Streaky, where it is not in measurable amount, and appears only as streaks in the sputum.

Small, where the quantity is less than 1 ounce.

Moderate, where the quantity ranges between 1 and 4 ounces.

Copious, where the quantity ranges between 4 and 10 ounces.

[1] *Gaz. d. Hop.*, 1865, No. 18.
[2] *Gaz. d. Hop.*, 1868, No. 61.
[3] *Gaz. hebdom.*, 1868, i. 146.
[4] *Trans. Path. Soc. Lond.*, vi. 75.
[5] *L'union méd.*, 1876, 931.
[6] *Phthisis*, p. 156.
[7] *Medic.*, p. 963.

Profuse, where the quantity is more than half a pint. Of this there are three forms:

(*a*) *Suffocative*, where the amount is so large as to suffocate the patient almost immediately.

(*b*) *Remittent* or *continued*, which may be (1) fatal after a time from exhaustion, (2) or gradually cease and end in recovery.

(*c*) *Intermittent*, the attacks being separated by intervals often of considerable duration.

The amount of blood brought up is usually small, under 2 or 3 ounces, and it may not be more than a few streaks in the sputum.

Walshe gives the average as less than half an ounce in 45 per cent., and the second Brompton Report as less than that amount in 69 per cent.

My own statistics show hæmoptysis absent in 30 per cent., streaky in 23 per cent., slight in 34 per cent., copious in 13 per cent.

From a comparison of various statistics I think the following averages may be taken as approximately correct:—

Streaky (no measurable amount), Small (less than 1 ounce),	. . . . .	50 per cent.
Moderate (from 1 to 4 ounces),	. . . . .	15 ,,
Copious (from 4 to 10 ounces), Profuse (above 10 ounces),	(*not fatal*, 8—*fatal*, 2) . .	10 ,,
Absent,	. . . . .	25 ,,

These figures show that it is in not more than about 10 per cent. of all cases that hæmoptysis becomes an urgent symptom and requires active treatment.

Even in profuse hæmoptysis the amount rarely exceeds half a pint in the twenty-four hours, but this amount may be brought up day after day for even several weeks together. In most cases the blood is brought up in small mouthfuls each time the patient coughs, so that, though the amount at any one time is small, it adds up to a considerable quanity in the course of the twenty-four hours. The largest amount I have actually seen brought up in twenty-four hours in a case that was not fatal by suffocation is 36 ounces. The blood may now and then pour out of the mouth in gushes, but such cases are rare (under 1 per cent.), and almost immediately fatal by suffocation.

The frequency of hæmoptysis in the different stages of the disease is fairly represented by the following figures (Reginald Thomson):—

1st stage, 20·7 per cent.; 2nd stage, 44 per cent.; 3rd stage, 35·6 per cent.

Except as a matter of general interest, such statistics have little practical importance, for the difficulty of determining the exact stage of the disease is considerable, and it often happens that profuse hæmoptysis, occurring in what appears to be an early stage, proves to be due to very chronic and long-standing disease. This subject has already been dealt with when discussing the question of *phthisis ab hæmoptœ*.

What may be generally stated is this—

1. That hæmoptysis occurring in early or recent phthisis is usually slight, and is not, as a rule, met with until the tubercular mischief has advanced to the stage of breaking down.
2. That hæmoptysis may be completely absent throughout in even very acute cases.
3. That profuse hæmoptysis is associated with chronic cavities, and occurs, therefore, in the older cases of phthisis; and
4. That when it is met with in what seems to be a recent case, it proves almost without exception to be connected with latent and perhaps undiagnosable chronic excavation.

As regards the diagnostic value of hæmoptysis, Louis' dictum may be accepted, viz., that idiopathic hæmoptysis, *i.e.*, hæmoptysis coming on without apparent cause, is presumptive evidence in favour of tubercle; for on examining a series of cases with reference to this point, he found, with hardly an exception, that those who spat blood either were or became phthisical. The difficulty, of course, lies in excluding the possibility of the blood coming from other sources, *e.g.*, the nose or pharynx. If this can be done the statement is approximately true.

Source.—The blood may come from the bronchi or from the pulmonary tissue, and the hæmoptysis is called bronchial or pulmonary respectively. It may be bronchial in origin, when the bronchi are the seat of tubercular ulceration, or if the cough be violent, when the small vessels in the bronchi give way as in other cases of violent expiratory effort. With these main exceptions the bleeding in phthisis is connected with lesions of the lung tissue, *i.e.*, it is pulmonary. It is possible, of course, that round developing tubercular lesions the congestion may be sufficient to cause extravasation of blood; but the congestion in phthisis can hardly ever be so extreme as is seen in acute pneumonia, and in that disease hæmoptysis in any quantity is very rare, while, on the other hand, the acute cases of phthisis are, as a rule, characterised by the entire absence of hæmoptysis. Hæmoptysis, therefore, with few exceptions, is to be referred to gross lesions of vessels in the lung; to erosion in early cases, and in the chronic to erosion or ulceration, or still more frequently to rupture of an aneurysm. The subject has been fully dealt with in the article on Hæmoptysis.

The results of hæmoptysis.—When small it has usually no influence at all upon the course of the disease; it is simply an accidental occurrence and of no special importance. Even when more abundant, it may be of importance only so far as it increases the debility and anæmia, but these are quickly recovered from. Not infrequently it appears actually to do good, and improvement seems to date from the bleeding.

It is not rare to find pain in the chest, which has been previously complained of, relieved by the bleeding, and even for the time entirely removed. The relation between pain and hæmorrhage is striking and interesting, and will be further referred to.

If the bleeding from the lung be free, the blood will tend to gravitate to the lower parts, and may give signs of its presence there by crepitation and altered breath-sounds. As a rule these physical signs rapidly clear up and no further changes occur, but occasionally the temperature rises and an attack of subacute pneumonia supervenes, but this, too, in time resolves and may entirely recover.

Lastly, hæmoptysis may be followed by a very rapid and acute development of tuberculosis. This is the explanation of most of the cases of "phthisis ab hæmoptœ"; but, as already stated, the result is due not to the blood, but to tubercle bacilli carried by it and disseminated throughout the lung.

Hæmoptysis tends to recur, but with great irregularity, so that the intervals between the attacks may be indefinite, sometimes even many years.

Thus I have seen a man who had his first attack of hæmoptysis at the age of 24, and that a profuse one, and none again until the age of 38, fourteen years later. Another man of 27 had had attacks of slight hæmoptysis for fourteen years at irregular intervals, and one very profuse one at the age of 21. He presented the signs of chronic phthisis at the left apex, with complementary hypertrophy of the whole right lung.

Still, profuse hæmoptysis may recur at very short intervals. A man of 27 had three distinct attacks of profuse and dangerous hæmoptysis in seven months; and another of about the same age, three similar attacks in six months. In an attack of profuse hæmoptysis the outbursts may

be curiously periodic. Thus I had one case under observation in which profuse hæmorrhage occurred for seven days almost regularly at midday and midnight, the patient losing about half a pint each time.

An attack of hæmoptysis in a person predisposed to it may be determined by various causes, especially by sudden effort or strain, *e.g.*, muscular exertion, coughing or other violent expiratory effort as well as by mental excitement, probably through its action on the heart. The physical examination of the chest should always be made with delicacy and care, when patients are spitting blood, or are liable to it, for an attack may be aggravated or even brought on by rough manipulation.

Sex.—Profuse hæmoptysis is no doubt commoner in men than in women. For this there are two reasons: (1) that men are more frequently the subjects of chronic phthisis, and (2) that they are more liable from their occupations and greater exposure to causes which may excite it.

It has been asserted that haemoptysis is affected by season, and that it is more common in the warm than in the cold months, but I do not think that statistics bear this out.

Age.—Hæmoptysis may occur at any age, just as tubercle may, but it is commonest, of course, at the period at which phthisis is most prevalent, viz., during early and middle adult life. It is rare at either extreme, partly because at the extremes of life phthisis is rare, and partly because, if phthisis develop, it tends to run an acute course. I have seen one instance in a man of 68.

Profuse and still more fatal hæmoptysis is very rare in early life, but it has been met with even in infants, and in such cases it is found to be associated with the same lesions in the lung as it is in the adult.

Hoffnung[1] records a fatal case in a child of 10 months, due to the rupture of a pulmonary aneurysm in a cavity which was traced back to measles. He gives a *resumé* of 10 other cases collected from literature.

In 4 connected with aneursym of the pulmonary artery.
,, 1 ,, ,, suppurating bronchial glands opening into the pulmonary artery.
,, 2 ,, ,, gangrene.
,, 1 ,, ,, thrombosis of the pulmonary artery.
,, 1 ,, ,, pulmonary apoplexy (*sic*).
,, 1 without any discoverable cause.

Powell[2] records a case fatal at 7 months; Revilliod[3] one at 16 months; Cholmeley[4] one at 20 months; Lebert[5] another at the age of 2 years.

I have seen one case in a child of 5, who was under treatment for what was thought to be simply broncho-pneumonia. No *p.m.* could be obtained.

Pollock records a case, not fatal, at the age of 18 months.

B. THE PHYSICAL SIGNS.

The physical signs vary with the amount and nature of the lesions in the lung, and in any given case they differ also in different parts of the chest, and even in the same part are subject to continual change as the disease progresses. Their importance lies in the fact that it is by their means only that we can ascertain what the lesions are.

As tubercles form in the lung they produce inflammatory reaction in the parts around them, *i.e.*, in the alveoli and in the walls of the small bronchi. The early signs of phthisis, therefore, are those of local congestion or local bronchitis; a little wheezing and fine crepitation, especially on deep inspiration or after coughing.

[1] *Virch. Jahrbuch*, 1885, ii. 584.
[2] *Path. Soc. Trans.*, xxii.
[3] *Virch. Jahresbericht*, 1887, ii. 713.
[4] *Path. Soc. Trans.*, xxxix. *Cf.* Etiol. *Phthisis in Children.*
[5] *Amst. Krankh.*, 188.

The signs of local bronchitis, if persistent, are always evidence of local disease, and when present at the apex are almost conclusive as to the existence of phthisis there. As the tubercles develop, patches of broncho-pneumonia form. So long as they are small and deep-seated in the lung, they produce no definite physical signs, but if they become confluent, and form larger masses, they yield the ordinary signs of consolidation.

These patches of broncho-pneumonia may resolve, and the signs due to them may therefore disappear, but if, as is usual, they persist, they become themselves tubercular, caseate (caseating broncho-pneumonia), and after a time degenerate or soften. When the softened parts communicate with a bronchus they are expectorated and a cavity is formed.

In all but the most acute cases attempts are made at repair, by the formation of fibrous tissue, which cicatrizes and leads to contraction of the affected parts.

As the lesions vary in different parts of the lung so do the physical signs, so that all the different groups of physical signs may be present in the same case in different parts of the chest; thus at the apex, where the process usually commences, there may be the signs of excavation and contraction; lower down those of advancing consolidation; and lower still the early signs of broncho-pneumonia and local congestion.

When we speak in any case of the physical signs of phthisis, we mean those present in the seat of most advanced disease, but the signs in the other parts are of great importance as indicating whether the disease is progressing or not.

The physical signs vary also according to the acuteness of the disease, its diffusion or localisation, and again according to the parts chiefly involved, viz., the air-tubes, lung-tissue, or the pleura.

Thus, in one of the acute forms, where there is much inflammatory reaction, and it is localised, the physical signs resemble closely those of acute pneumonia; in another, where the disease is more diffuse, those of acute bronchitis or broncho-pneumonia. In nearly all cases the apex is attacked either first of all, or else most extensively, and this is an important factor in diagnosis, for physical signs which elsewhere might admit of other interpretation, at the apex may be conclusive of phthisis.

The pleura is always involved sooner or later, but it may be the first part to yield physical signs—either those of a dry pleurisy or of an effusion. In the case of dry pleurisy the localisation and distribution are of great diagnostic importance. If it be limited to the apex, and not due to croupous pneumonia, it is almost conclusive as to the existence of tuberculosis. So it is also, if it be very widespread over one lung, or still more if it be bilateral, even though it be confined to the lower parts. Speaking generally, double pleurisy is due to the same lesion exciting it on both sides of the chest, and if not malignant disease, there is practically under ordinary circumstances little else for this to be but tubercle.

When effusion takes place, even if the original cause be at the apex, the fluid will gravitate to the lower parts. Tubercle is a very common cause of pleuritic effusion, but there is nothing in the seat of the effusion itself, nor in its character, to determine the diagnosis as to its cause.

In the healthy chest the physical signs are remarkably symmetrical, and it is the disappearance of this symmetry, or, in other words, the development of a difference in the physical signs on the two sides of the chest, that yields the most important evidence of disease. Hence it follows that in examining the chest for the signs of phthisis the corresponding parts on the two sides must be carefully compared with each other. It is not a difference between the physical signs in the case under examination compared with some ideal average or standard, but

an actual difference between the two sides of the particular chest in question, a difference which is usually easy enough to determine, even when very slight. The only fact to be borne in mind is, that at the right apex in the healthy chest the expiration may be slightly audible, and the vocal vibrations and vocal resonance a little increased, as compared with the left side. This is a physiological difference, and even so not always present. If the same difference were reversed, *i.e.*, if it were on the left side that the exaggerated physical signs were present, they would indicate disease.

Auscultation.—The physical signs yielded by auscultation are not only the earliest to be detected, but remain throughout the disease the most important, so that it is natural to consider them first.

In the healthy chest, expiration is either not audible at all, or if so, hardly at all, and then only at the commencement. In early phthisis one of the first changes is that expiration becomes audible and prolonged. At the same time inspiration becomes somewhat louder and harsher, so that the soft, blowing, vesicular murmur of the healthy lung disappears, and its place is taken by the altered breathing sounds described.

The inspiration is often wavy or interrupted (saccadée, entre-coupée).

This is not pathognomonic of phthisis, for it may occur when the lung is quite healthy in patients, who are not, and do not become, phthisical. It is very variable, being present at one time and not at another, appearing and disappearing quite irregularly. This interrupted breathing is due to interference with the entry of air, owing usually to some secretion in the tubes, but it appears to be also sometimes muscular in origin, *i.e.*, due to irregular muscular action.

As the disease progresses, the expiration becomes longer and louder, until at last it is equal in length and loudness to inspiration, and both sounds acquire a harsh character. This is what is usually called bronchial breathing.

The alteration in the length and characters of the breath sounds progresses as consolidation advances, even up to true bronchial breathing; but it does not follow that these changes are associated with an increase in loudness, for it often happens that, owing to the diminished entry of air into the affected lung, the breathing sounds are actually less loud on the affected than on the sound side, though the characteristic changes, viz., the prolongation of expiration and the harsh character of both sounds, may be well marked. This may occur even with bronchial breathing, and it is this which is meant when the bronchial breathing is described as weak.

When cavities are present, and lie near the surface, the breath sounds have added to them the hollow sound which is described by the term cavernous or amphoric.

When, owing to some affection of the larynx, there is loud laryngeal stridor, the interpretation of the auscultation sounds over the chest becomes extremely difficult, for it is impossible to say how much is produced *in situ* and how much is propagated from the larynx.

I have, under such circumstances, seen very extensive mischief, even a large cavity, completely missed, even after careful and repeated examination for that purpose by experienced observers.

In all cases the effect of secretion in the tubes must be borne in mind, for where the tubes are plugged, so that no air enters the affected parts of the lung, no breath sounds may be heard at all; yet when the tubes have been cleared by coughing and expectoration, the signs of consolidation or cavity, as the case may be, become evident. It is a good rule, therefore, to make the patient breathe deeply several times and cough once or twice, and then immediately afterwards to examine the chest again.

Change of position sometimes has the same effect. Where there is a large cavity containing much secretion, in one position, *e.g.*, the recumbent, the secretion may cover the mouth of the main bronchus communicating with the cavity, and so no breath sounds be heard. Change of position, *e.g.*, sitting up, may change the position of the fluid and leave the mouth of the bronchus free, and so the breath sounds may become audible. Such changes are most frequently seen with cavities at the base, but I have seen them also at the apex, and that in an early case in which there was no other evidence of a large cavity; still the explanation was, I believe, the same, viz., that in one position the tubes were occluded and in the other free.

Sometimes after cough a high-pitched, sucking, inspiratory sound is heard (*post-tussic suction*), due to the sucking in of air into the cavity, as it is suddenly relieved from compression after the cough is over.

Crepitation.—Crepitation of all kinds may be met with in phthisis; fine-hair, fine, medium, large, gurgling, bubbling, etc. In the earliest stage it may be very fine and due to the opening out of collapsed air vesicles, or to a little fluid in the air-tubes. Fine crepitation cannot be distinguished by the ear from some forms of pleuritic friction, but the distinction is of no importance in the case of phthisis, for at the apex its meaning in either case is the same, viz., tuberculosis. Crepitation is, as a rule, due to the bursting of air-bubbles in fluid either in the air-tubes or in cavities. Its character will depend upon the size of the bubbles and of the tubes or cavities in which it occurs, and upon the modifications in sound produced by the changes in the lung tissue around. In the early stage, where the consolidation is dense, the crepitation is often clear, sharp, and crackling. It is to this form that the name subcrepitant has been given, and it is sometimes mentioned as if it were pathognomonic of phthisis; this it is not, for the same kind of crepitation is met with in other affections of the lung where the conditions are similar, *e.g.*, in broncho-pneumonia, and occasionally in croupous pneumonia. When cavities are present it is often gurgling or bubbling, and may acquire a ringing or metallic character.

The importance of crepitation is that it is the chief indication of softening or breaking down.

In early stages crepitation may not be audible with ordinary breathing, but only after deep inspiration or after a good cough. By-and-bye it becomes audible with both inspiration and expiration and on ordinary breathing.

It has been asserted that with hæmoptysis the crepitation is peculiar and pathognomonic. This is an error, for though crepitation heard during hæmoptysis at the bases, or in places where it was not heard before, is presumably due to blood in the tubes, it has no special characters by which it can be diagnosed.

In cases where there is rapid consolidation, and no breaking down, *i.e.*, in certain acute cases of phthisis, crepitation may be absent throughout, but this is quite an exception.

Vocal Resonance.—The voice sounds, like the breath sounds, undergo gradual increase as the consolidation advances, until they become intense, and are described by the term bronchophony. They may be transmitted with unusual clearness and directness to the ear, and are then termed *pectoriloquy*, and these changes may be evident both with the ordinary voice or with the whisper, when they are called *whispering bronchophony* or *pectoriloquy*. With large cavities the voice sounds, like the breath sounds, may acquire a hollow amphoric character.

Bronchophony and pectoriloquy are often very local, and, like the other auscultation phenomena, vary greatly and irregularly in different parts of the chest, and in the same parts at different times.

There are two other modifications of vocal resonance which deserve mention, both of which may be termed *autophony*. In the one the patient himself feels the increased vibrations of his own voice, and refers them correctly to the seat of disease. I have had a patient complain of this as a great trouble and inconvenience to him in talking. In the other the auscultator hears his own voice with undue loudness if he speaks while his ear is on the chest or stethoscope. In this case his own vocal resonance is, as it were, echoed or reflected back to him, or perhaps in both cases the effect may be a form of consonance. I have, however, only seen it with consolidation, and never with a large cavity or with pleurisy.

Palpation.—Of the physical signs on palpation little need be said. The vocal fremitus is increased like the vocal resonance, and usually in the same degree; but not always, for it may happen that the vocal resonance is distinctly increased, and has peculiar characters, when the vocal vibrations are hardly to be felt, or if at all, not in more than normal degree.

When the consolidation is superficial and extensive, the sense of resistance is greatly increased, and even in the early stages, when percussion is not greatly impaired, the altered resistance felt on percussion (*percutory palpation* or *palpatory percussion*) is very marked and a great aid to the ear; while in advanced cases the want of vibration on percussion can be felt, as plainly as it is heard.

Percussion.—In the very early stage, even when auscultation yields suspicious signs, there may be no impairment of resonance to be detected on percussion. On the contrary, the resonance may even be tympanitic, and differ by way of excess rather than of defect. This hyper-resonance is no doubt the result either of an actually emphysematous condition of the parts of the lung surrounding the tubercular foci, or of a condition of relaxation of the lung tissue due to impairment of its nutrition, and similar to that observed not infrequently with pneumonia (Skodaic resonance); thus it may be hyper-resonant in front, with dulness behind; and at the apex this may at times be misleading. Before long, and it may be very early indeed in the case, the percussion becomes impaired on the clavicle, or just above or below it, the note being at first flatter and somewhat higher pitched on the affected side. As consolidation advances the note becomes more and more impaired up to distinct dulness. Absolute dulness or complete non-resonance, such as is common over pleuritic effusions, is rarely, if ever, met with in phthisis, no doubt owing to the fact that the consolidation is rarely homogeneous and complete, but contains either patches of air-containing lung tissue or cavities.

Over cavities the percussion note may be somewhat resonant, or the note which had been dull may become less so. A tympanitic note or marked hyper-resonance has been described, but it is of the rarest possible occurrence. Even when the note is resonant it is almost invariably flat, or, as it has been termed, "boxy," *i.e.*, a note such as we obtain when percussing an air-containing cavity with thick walls.

Variations in the percussion note over cavities, which are said to be obtained according as the mouth is open or closed, are more often described than actually seen, and it is very rarely that such changes can be demonstrated, unless the cavities are of large size, superficially placed, and in free communication with the air-tubes.

Inspection.—Inspection usually reveals, even quite early in the disease, changes both in movement and in shape, though in very early cases they may be so slight as to be easily overlooked.

Defects in movement are often to be observed before any change in shape occurs. They may be detected both by the eye and hand, often more easily by the latter.

To examine the movements, the hands should be placed symmetrically on the two apices, either in front or from behind. In the latter case the thumbs should be placed close together on the vertebræ, and the fingers spread out over the clavicles, upon the upper intercostal spaces in front. This method is especially convenient for women and children.

The defects of movement are due to three causes—(1) deficient entry of air from obstruction to the air-tubes; (2) diminished expansion of the lung, owing

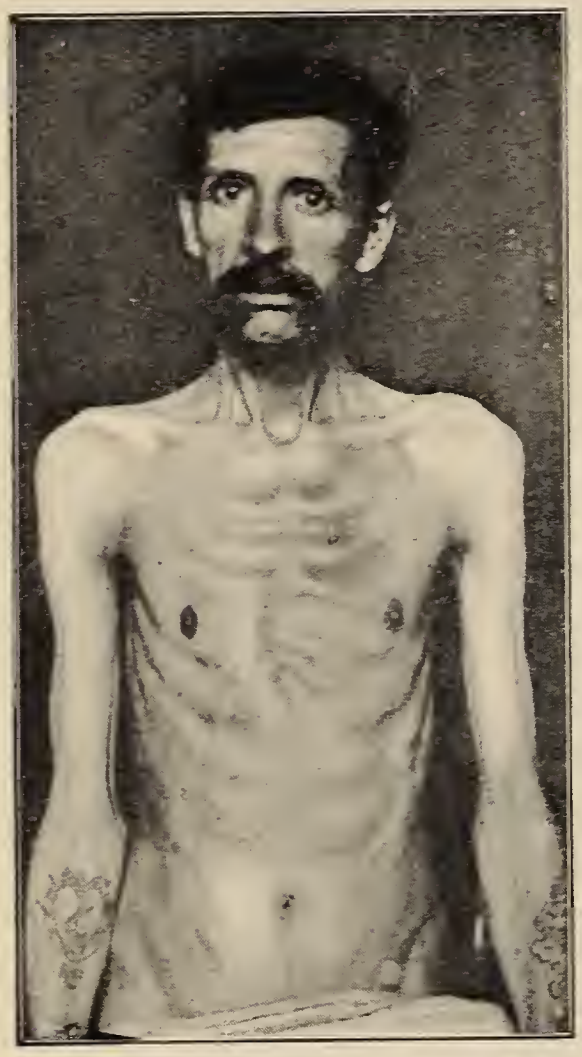


Fig. 130.

Photographs from an advanced case of phthisis. *a*, shows the long, narrow, flat shape.

Fig. 131.

b, The dropping forward of the shoulders and depression of the intercostal spaces.

to consolidation, or to fibroid change in the lung or pleura; (3) pain on breathing.

At the same time it must be noted, that the movements may not be affected, even when the other physical signs are well-marked.

Change in shape may not be obvious in early stages, but as the disease progresses, it becomes marked, owing to the contraction in the lung or pleura, or in both. It is most marked in the upper parts of the chest, and may be so extreme as to cause the shoulder to fall forward, and the scapula to tilt out from the back, thus producing the form of chest described as the *alar* or *pterygoid*.

On the other hand, there may be little or no change in shape, even with considerable and long-standing disease of the lung. This is usually met with

where the changes in the lung are unilateral, so that, as the one lung has contracted, the other, remaining healthy, has increased in size in the same degree. The deformity that results is internal, not external, and the form of the chest may remain normal. This condition of contraction of one lung with complementary hypertrophy of the other will be further considered later.

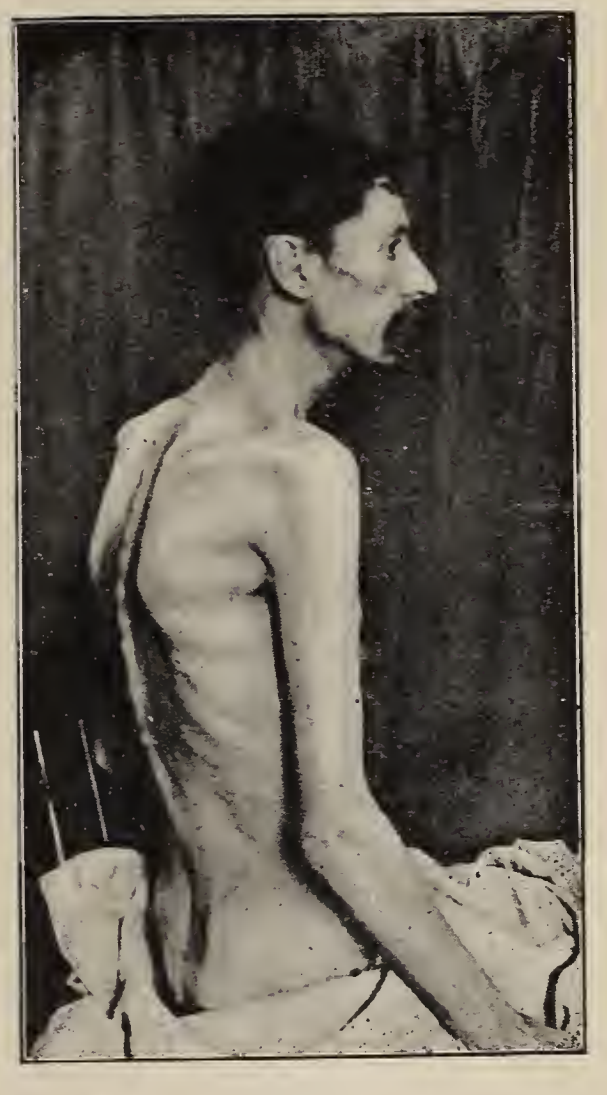


Fig. 132.

c, The tilting of the scapulæ.

Dilated Superficial Veins.—The subcutaneous veins over the upper intercostal spaces are often unduly distinct and dilated. This is associated with adhesions and chronic pleural thickening in the parts beneath, and is due, no doubt, to the pinching of the branches of the intercostal veins on the inside of the thoracic walls, the blood which would normally pass through them being returned by anastomotic channels through the superficial veins. There is, I believe, no direct evidence that there is any anastomosis between the vessels of the lung and those of the thoracic walls through the pleural adhesions.

Cavities. — We know that, wherever tubercular consolidation breaks down, cavities are formed; but the diagnosis of cavities is more often a matter of inference from our pathological knowledge than of direct demonstration by physical signs. In order that the cavity should be recognised by physical signs, it must have reached a certain size, be in free communication with the air-tubes, and be not far removed from the surface. Even so, very large cavities may not be recognised, for they may yield absolutely no characteristic physical signs, and sometimes signs apparently conclusive of cavity may be present, and yet no cavity exist.

The characteristic signs of cavity are amphoric or cavernous breathing, amphoric or metallic echo, and gurgling crepitation; but the largest cavities may yield no conclusive physical signs if the tubes are occluded or the cavities filled with secretion.

As there are several points to be noted about the physical signs of cavity, it will be well to consider them in order.

Inspection.—It is the rule over cavities for the side to be flattened and the movements to be impaired, but there may be no defect of either movement or shape; on the contrary, the movements may be free, and the part even more prominent than normal. Such conditions are, of course, very rare.

Percussion is usually impaired or dull, but it may be boxy; tympanitic it hardly ever is. The note is said to change according as the mouth is open or shut, but, as it is necessary for this that the cavity should be superficial and of considerable size, and in free communication with large air-tubes, it is rarely observed.

Bruit-de-pot-fêlé (*Cracked-pot sound*) is of no value as a cavity sign. It may be found over a cavity, but it is not uncommon where there is none, as, for instance, over relaxed lung tissue in front of consolidation, or even over pleuritic effusion. The best instance of it I have ever seen occurred over an enlarged heart where the lung was perfectly healthy.

It is a very transient phenomenon. It is best elicited by sharp percussion when the patient is breathing out and has the mouth wide open.

The production of this sound depends a good deal upon the elasticity of the chest walls. Thus it is often easily obtained in children with perfectly healthy lungs, when they are crying with the mouth wide open.

Fagge describes a case in which he obtained the bruit-de-pot-fêlé over an abscess in the chest wall due to the pointing of a pyo-pneumothorax.

Auscultation yields the most conclusive signs of cavity, for both the voice- and breath-sounds may acquire cavernous or amphoric characters; still their absence is not conclusive as to the absence of cavity, and amphoric sounds may be present where no cavity exists. I have seen well-marked amphoric breathing with a large pleuritic effusion over the upper part of the affected side, and I have seen it appear there immediately after paracentesis, when not present before the operation. I have seen also at both bases such distinct amphoric breathing and gurgling crepitation as to warrant the diagnosis of cavity, and yet *post-mortem* nothing was found but a condition of solid œdema, the remains of an imperfectly resolved pneumonia. This occurred in a patient who had had an acute illness about six weeks before she came under my observation. She presented all the appearances of acute tuberculosis, and died after eight or nine weeks' illness.

Helmholtz's resonators have been suggested for the purpose of determining the size of cavities, but the conditions in cavities are rarely such as to yield consonance, and if they do, no conclusion as to the size of the cavity can be drawn; in fact, if this cannot be determined by the other methods of physical examination, a resonator will not do it.

Whispering pectoriloquy is frequently heard over cavities, but is of no diagnostic value.

Metallic echo (*Gutta cadens*), a ringing sound like that produced by the falling of a drop of water in a grotto, is characteristic, but is rarely heard.

Both crepitation and metallic echo may be produced by the movements of the heart in cavities placed near it, as well as by breathing.

Metamorphosing respiration, probably the same as Laennec's *souffle voilé*, is the name given to the breathing sounds which alter their character as inspiration proceeds, being at first indefinite and then becoming bronchial or amphoric, as the case may be. It is not characteristic of cavity, for it occurs also with solid lung. Its meaning is the same in either case, viz., that the air-tubes are not freely patent and only become so as inspiration proceeds.

When a cavity is very large, as where the whole or greater part of one lung is excavated, the physical signs may resemble those of pneumothorax, for the percussion note may be somewhat hyper-resonant, the breathing may be amphoric, and there may even be succussion if the cavity contain fluid. Still mistakes can hardly be made with proper care, for the note is rarely tympanitic, but almost always of a flattened boxy character; the resonance does not extend, as in pneumothorax, right down to the costal arch; the side is not distended, but somewhat contracted, and, if the heart be displaced, it is towards the excavated side, and not to the opposite side. I have seen several cases of this kind, and in some the diagnosis had been made of pneumothorax, but the mistake might have been avoided by attention to the points mentioned.

The physical signs of cavity may after a time disappear, but this is no proof that the cavity has become obliterated. I believe complete obliteration can never occur in any cavity which has ever been large enough to have been diagnosed. It is very difficult in any case, and impossible in most, to diagnose even the contraction or diminution in size of a cavity, though the claim to have done this successfully has been sometimes made.

I have certainly seen, in a patient with chronic excavation of one apex, the upper parts of that side fall in, and in so far the cavity probably became smaller, but the effect was due to the contraction of chronic pleurisy rather than to any primary contraction of the cavity. We know, of course, from pathological evidence that cavities do contract, but it is quite another matter to determine this by physical signs.

It is very rare for a cavity to extend beyond the lung. It often reaches to the pleura, but very rarely beyond, for the thick fibrous tissue there usually prevents its further extension.

Occasionally, however, as the result of suppuration or tubercular disintegration, the thickened pleura may be pierced, and the thoracic walls involved. An abscess may thus be formed which may ultimately burst through the skin, and produce an external fistula.

A case of this kind is recorded by Snell,[1] and another is described in the *Berl. klin. Woch.*, 1892, No. 20.

I remember one case in which a patient, when smoking, could expel tobacco smoke in a good stream through such an opening in his side.

Sometimes the abscess which forms contains air, and yields a resonant note, but it also happens that without suppuration, subcutaneous emphysema may develop and spread widely over the side of the chest.

Cases of this kind are described by Fagge[2] and Fraentzel.[3]

When a cavity exists near the heart, on the one hand the heart sounds may be modified by it and become exaggerated, ringing or amphoric in character; on the other hand the movements of the heart may produce peculiar sounds in the cavity, *e.g.*, crepitation, murmurs, or even succussion. In this connection may be mentioned those remarkable cases in which the heart sounds are so exaggerated as to be propagated to a distance even of several feet from the patient. Fagge[4] and Taylor[5] have both recorded cases of this kind. In each instance the propagation of the sound could be stopped by covering the mouth and nostrils.

Röntgen Rays.—Attempts are being made to utilise the Röntgen Rays for the diagnosis of lung lesions, especially those of phthisis, but so far with only very limited success.

The recent work of Dr. Hugh Walsham seems, however, to be of a more promising character in this respect. Still, however useful the examination of the chest by Röntgen Rays may ultimately prove to be, it is never likely to be more than auxiliary, or to become a substitute for the ordinary methods of physical examination.

The Physical Signs in other parts of the Chest.—The physical signs in different parts of the chest vary and change even in the same part as the disease progresses, but beyond the physical signs, which are due directly to the tubercular lesions, others occur which are the result of secondary changes in the parts not yet involved. They are of two kinds, viz., collateral congestion and collateral emphysema or compensatory hypertrophy.

The *collateral hyperæmia*, if it produces any signs at all, yields those of congestion, *i.e.*, of bronchitis, either localised or general. When localised, it is most marked in the immediate vicinity of the tubercular lesion, and often indicates the commencement of fresh tubercular foci.

Collateral emphysema and *complementary hypertrophy* are of two kinds. The one is transient, probably the result of the altered nutrition in the parts adjacent to the active tuberculosis, and in all respects similar to that met with in broncho-pneumonia. The other is permanent, and more nearly allied to hypertrophy than to emphysema. It is that persistent dilatation of the healthy parts of the lungs which follows upon the contraction of the diseased parts. It may be quite local; for instance, in chronic phthisis of one apex it may involve only the unaffected parts of the rest of the upper lobe, and this may be quite sufficient for complete compensation, so that, although the apex may be greatly contracted, the general size of the lung may not be altered; in other words, the liver dulness on the right side, or the stomach resonance and cardiac dulness on the left, may be in their normal position.

[1] *Lancet*, Sept. 30, 1893.
[2] *L.c.*, p. 966.
[3] Ziemssen, iv., Hft. ii., p. 542, 2nd ed.
[4] *L.c.*
[5] *Clin. Soc. Trans.*, 1880.

If parts of both lungs be contracted, and compensation be not complete, the boundaries of the lungs will show this; the liver dulness will rise to the fifth rib or higher, the cardiac dulness will be increased upwards, and the stomach resonance likewise.

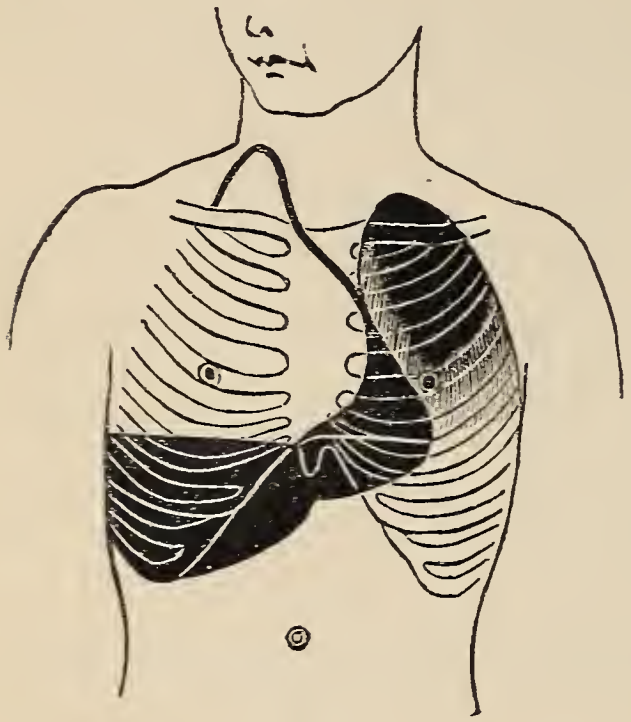


Fig. 133.

Diagram showing the displacement of the middle boundaries of the lung, with contraction of left lung and complementary emphysema of the right.

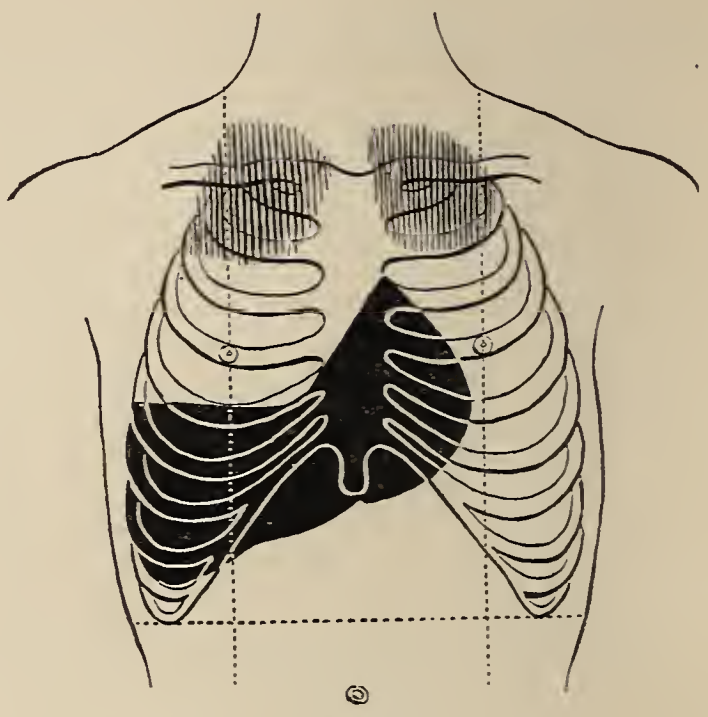


Fig. 134.

Diagram to show the displacement of organs, with contraction of both lungs.

Complementary Hypertrophy.—If one lung only be diseased, and the opposite lung remain healthy, it may hypertrophy, and may in the end reach such a size that no external evidence of the contraction is visible. This leads to the most extreme dislocation of boundaries, for as the affected lung contracts, the mediastinum moves over to the contracted side, taking with it the heart and the opposite lung, which may then, instead of reaching only to the middle line of the sternum, extend even as much as 2 inches at the level of the third rib into the contracted side. In these cases the liver dulness usually remains in its proper place, but the change in position of the lungs beneath the sternum can be easily made out by percussion. I have frequently verified the surface markings made during life, with the position of the lungs after death, and found them to tally exactly.

It is in such cases as these that no external evidence of the contraction may be evident.

This compensatory enlargement of the opposite lung is of clinical value in prognosis, for it does not take place unless the opposite lung is healthy, and conversely, when it occurs it may be taken as evidence that the lung is healthy. The condition is met with usually in chronic cases of phthisis, but I have seen it develop rapidly in subacute cases, and it has always appeared to me a favourable sign.

The physical signs vary with the progress the disease has made, and phthisis has accordingly been divided into stages.

The first stage is that of early consolidation without obvious cavities; the physical signs of this stage are more or less impairment of percussion, with more or less exaggeration and alteration of the voice- and breath-sounds.

The second is that of softening or breaking down, and commencing excavation, as shown by the various kinds of crepitation.

The third stage is that of well-marked excavation, yielding the signs of cavity.

As a purely pathological classification it is admissible, but when applied to the clinical conditions during life it becomes confusing and inaccurate; for it rests on the assumption that there is close correspondence between the clinical condition of the patient and the amount of pathological change, and though there may be a rough correspondence between them, there is, on the other hand, frequently none. It does not by any means follow that a patient with marked excavation and extreme physical signs is clinically in the third or final stage of the disease, and, therefore, likely soon to die. On the contrary, there may be the most marked disproportion between the amount of mischief in the lung, as shown by the physical signs, and the general condition of the patient, which, so far from being grave, may be that of moderate or even good health, while on the other hand, a patient in the first stage may be so ill that it is clear he has but little time to live.

The physical signs indicate only the amount and kind of lesion present at the time in the lung, and these may have been produced long ago, and be at the time of examination in a stationary condition.

What we require to know for the purposes of prognosis is, Whether the disease is active, *i.e.*, progressive, or not? This information is supplied not by the physical signs so much as by the constitutional condition. The only clinical classification which is of any value is that which is based upon the latter, viz., the classification into acute, subacute and chronic, implying thereby that the disease is active and rapidly progressing, not very rapidly progressing, or progressing very slowly or not at all. In other words, it is not the physical signs that are of importance so much as the constitutional signs or clinical condition.

Phthisis may be arrested at any stage. When arrested the physical signs, or any of them, may persist, showing the mischief which has been done but not when it was done, or whether it be progressing or not. After remaining stationary, perhaps for years, the disease may again break out into activity, and recrudesce.

C. SPECIAL SYMPTOMS.

Of the special symptoms, those connected with the respiratory system are of prime importance.

Respiratory Symptoms.—The breathing is almost always accelerated to some extent. Its rate is easily increased by any extra demand made upon the respiratory organs, *e.g.*, movement, talking, coughing, or excitement. In many cases it is only under these conditions that any complaint of shortness of breath is made. In very chronic cases, especially where the rest of the lungs have undergone compensatory hypertrophy, there may be no shortness of breath even on exertion.

I have known an instance in a boy at a large public school, one of whose lungs was much damaged, who was the fastest short-distance runner of his time in the school.

The *acceleration of respiration* is, no doubt, chiefly physiological, an attempt being made to compensate for the diminished respiratory area by more rapid respiratory movements.

Both the acceleration of breathing and the dyspnœa vary to some extent with the amount of disease in the lung and with the fever, but chiefly with the rate of development of the disease, the most acute cases exhibiting symptoms of

distress out of all proportion to the amount of disease present, as indicated by the physical signs; for in the lungs, as in other parts, more marked symptoms are produced by a small amount of disease which has developed rapidly, than by a much larger amount which has taken some time to develop. This is no doubt owing in great part to the nutritive and circulatory disturbances in the rest of the lungs, which have not had sufficient time to adjust themselves to the change.

The *dyspnœa* may arise quite suddenly.

A man of 32 came to me saying that he was asthmatic, *i.e.*, he complained of periodical attacks of severe dyspnœa. He had a large cavity at his right apex, over which the physical signs varied greatly at different times. His dyspnœa was evidently due to the periodical discharge of the contents of the cavity into the tubes. The dyspnœa then produced was sometimes extreme and very alarming.

In the same way, profuse hæmoptysis may cause severe or even fatal dyspnœa.

But besides these groups of cases, there is another in which dyspnœa suddenly becomes severe, and for which it is difficult to assign a cause, even after *post-mortem* examination. It is in this way that phthisical patients sometimes die. In some of these cases the dyspnœa is due to thrombosis either in the heart or large pulmonary vessels; but in others, no cause in the heart or vessels can be found, and the explanation is still to seek.

Lebert found dyspnœa present in marked degree in 20 per cent. only of his cases, and it was entirely absent in 30 per cent.

The existing shortness of breath due to the local mischief will, of course, be greatly aggravated by any complication which may arise. Of these, the commonest are bronchitis, pain on breathing, pleuritic effusion, or acute pneumonia; besides these there are certain local causes, for instance, obstruction of the tubes by secretion suddenly discharged into them from cavities, or pressure on the trachea or main bronchi by enlarged bronchial glands.

Cyanosis is rare to any marked degree, except in the later stages or in acute forms of the disease. In most other cases the occurrence of cyanosis would indicate some complication, *e.g.*, bronchitis, pneumothorax, pleurisy, or miliary tuberculosis, etc.

Pain in the chest is frequent, but often transient. It may depend upon local pleurisy, but is often muscular, and caused by the straining of coughing.

Muscular pain may be felt in any part of the chest, but a very common place for it is at the attachment of the abdominal muscles along the costal arch, and it is then often symmetrical.

The chest is often tender on percussion, especially when pleurisy is present, but frequently there is general superficial hyperæsthesia rather than localised pain. This depends upon the general condition. It may be so severe as to render examination of the chest impossible for the time, but it readily yields to the application of tincture or liniment of aconite, and once removed may not return, at any rate for some time.

Pain sometimes stands in a peculiar relation to hæmoptysis, being not infrequently complained of in that part of the chest from which the blood seems to come.

This pain may be felt for some days before the hæmoptysis commences, and often immediately and completely disappears after the blood-spitting.

In one instance of this kind the pain preceded the hæmoptysis so constantly that the patient came to me to have the hæmoptysis, which she knew was imminent, stopped; and I have known other instances in which the hæmoptysis was welcomed as promising relief from the pain.

The hæmoptysis may be sometimes checked by the application of a leech or two to the place it is referred to.

This association between pain and hæmorrhage is seen also in gastric ulceration, in cerebral hæmorrhage, in menstruation, and occasionally in superficial wounds.

It is most remarkable in connection with chronic gastric ulceration, for here the ulcer is present before and after the bleeding, and yet the relief given by it to the pain is often complete for the time.

This question will be found more fully considered in the St. Bartholomew's Hospital Reports for 1882.

Circulatory System.—The *pulse* varies with the constitutional condition of the patient. Where the constitutional symptoms are well marked, the pulse rate is increased, the tension low, and the wave ill-sustained.

Bristowe states that the pulse is increased in hardness at first, becoming only diminished in force and fulness as the disease progresses. I cannot agree with this statement, for it is the rule, even in the most acute cases, for the pulse to be soft and feeble.

The acceleration of the pulse varies with the amount of fever, and though the pulse may be slow when the temperature is high, as in other cases of fever, this occurs but rarely. As in other forms of debility, the rate is easily accelerated by exertion and excitement.

The *pulse-respiration ratio* is but little altered in phthisis unless there be acute inflammatory mischief, or very widespread and rapidly-developing disease in the lung, or painful pleurisy.

The pulse, respiration, and temperature stand, as a rule, in the usual relation which they hold to one another in fever, and any departure from this relation may be of important significance. Thus a very rapid respiration-rate may indicate fresh or extensive mischief, and a very rapid pulse or an unusually slow one may indicate either cardiac irritability or cardiac failure.

The *heart*, beyond the acceleration of its action dependent upon the fever, is, in the majority of cases of phthisis, but little affected. It may happen, as in other fevers, that cardiac asthenia is an early and prominent symptom, the action being weak and the sounds, especially the first, feeble. This is probably toxic in origin, but it is a condition hardly ever met with except in very acute cases.

Organic disease of the heart and phthisis are occasionally associated, but the association is accidental, as is the case also where the pericardium is affected, except in the rare instances in which it is involved by direct extension from the pleura or lung, or has itself become tubercular.

The heart may share in the general wasting of the body, and be found much below the average weight; still this does not appear to occur so often as has been sometimes stated.

In 64 cases in which the weight of the heart was taken it was found to average 9½ ounces in the males and 7½ ounces in the females; in other words, to be about normal. It was below the average in 15 per cent., the heart weighing between 5 and 6 ounces in 12 per cent. of these, while in the remaining 3 per cent. it was still smaller. The smallest hearts in the series weighed 3 ounces and 3½ ounces respectively in two females, each aged 20 years, and 3½ ounces in a boy of 12 years of age.

Lebert found the heart markedly atrophied also in about 16 per cent. only.

Wasting of the heart, though not constant in phthisis, nor peculiar to it—for it is met with in other wasting diseases, such as cancer—is hardly ever found in any other chest disease except phthisis.

On the other hand, dilatation of the right side, which is common in many other chest affections, *e.g.*, in bronchitis and emphysema, is not common in phthisis. In bronchitis and pneumonia it is this dilatation and failure of the right heart which is so serious and so often ushers in death, while in phthisis death is more often the result of general asthenia than of cardiac failure.

As in other forms of anæmia, the heart may become temporarily dilated after exertion either on the left side only or as a whole; transient murmurs also may be present, hæmic in origin and heard at the usual places; but it is remarkable that in phthisis, though anæmia is so marked and constant a symptom, hæmic murmurs should be relatively less frequent than in idiopathic and other forms of anæmia.

The pulmonary second sound is usually accentuated and often reduplicated. The closure of the pulmonary valves may be often felt in the second left intercostal space, where also systolic as well as diastolic impulse may be seen and felt.

Valve-shock.—This is due to the uncovering of the pulmonary artery by the contraction of the parts of the lung which normally lie over it.

When the lung is consolidated near the heart, the heart sounds will be exaggerated as well as loudly and widely propagated, and sometimes systolic murmurs may be heard over the aortic or pulmonary artery, which have been referred to compression or pinching of the vessel by the consolidated lung.

If a cavity be near, the heart sounds may be altered in tone and acquire a ringing or amphoric character, or the movements of the heart may cause crepitation or even succussion and sometimes murmurs in the cavity, and these sounds may be heard even at a considerable distance from the patient.

Murmurs.—When the heart is greatly displaced in consequence of contraction in the lung, murmurs have been described as due to this displacement. This may be so, but I have never seen it, and such murmurs are certainly very rare with the much greater displacements which result from pleuritic effusions and pneumothorax. When the displacement of the heart is extreme, it may simulate aneurysm, as in a case recorded by Brackenbridge,[1] where the heart was high up on the right side.

Murmurs are occasionally heard in other parts of the chest not in connection with the heart and great vessels. They are almost always systolic in time, soft and blowing in character, and vary greatly with respiration. Apart from the subclavian murmur, which is the commonest of them all, these murmurs are, according to my experience, chiefly heard behind between the shoulders or in the lower parts of the lungs. Their pathology is very obscure. In some instances they may be vascular, and be caused by a vessel being pinched by some chronic fibroid change in the lung, or by being pressed upon by enlarged glands. In other cases, I think, they are not vascular at all, but *pulmonary* or *pneumonic*, and caused by air being forced out of lung tissue, which may be healthy, or out of a cavity, by the systolic distension of a large vessel. One or two remarkable instances are recorded in which, in the parts where the murmur had been heard, a cavity was found containing an aneurysm of the pulmonary artery to which the murmur was referred.[2]

The *subclavian murmur* is heard beneath the outer third of the clavicle. It is systolic in time, usually soft and blowing, but often harsh and loud, and sometimes heard only on inspiration. When occurring in phthisis it has been attributed to compression of the vessel during inspiration by adhesions resulting from chronic change in the apex of the lung or pleura, as the vessel crosses the first rib. It is rare in phthisis, and by no means peculiar to it, for it is often heard in perfectly healthy persons. One peculiarity it has, even in phthisis, and that is that it is not always present; in other words, it is inconstant, and very variable, facts which oppose the explanation usually given, as does also the further fact that such murmurs are rare in women. Sometimes the murmur is no doubt produced by the pressure of the stethoscope, but it often depends upon the position of the arms, and is then the result of muscular pressure upon the vessel. Besides these, there is a group of cases in which the subclavian artery undergoes temporary dilatation. The swelling may be quite obvious to the eye and hand, the pulsation may be abnormally increased, and a distinct murmur be heard on auscultation, and even the superficial veins over it be dilated. In fact, the condition may resemble that of a subclavian aneurysm, with which I have known it confounded, but with this difference, that all the phenomena may rapidly disappear and as rapidly return. The condition is, no doubt, closely allied to that of abdominal pulsation, and is sometimes met with in association with it. At any rate it is by no means uncommon, though not very frequently recorded.[3]

[1] *Lancet*, 1880, i. 80.
[2] Gerhardt, *Lehrb.*, Ed. iii., pp. 218, 270.
[3] *St. Barthol. Hosp. Rep.*, vol. xvi. 119.

Digestive System.—Digestive disturbance is no necessary part of phthisis, but it stands in close relation to the constitutional signs, being, as a rule, considerable where they are well marked. Thus in acute, rapidly-advancing cases the tongue is usually covered with a creamy fur; there is often much thirst, and appetite is lost; vomiting is frequent; the bowels are irregular, sometimes constipated, sometimes loose; there is often pain in the stomach after food, and intestinal colic.

In the subacute or chronic cases there may be little or no gastro-intestinal disturbance unless there be some lesion in the intestines, such as tubercular ulceration or amyloid disease. Conversely, the occurrence of digestive disturbance in any given case is a sign of grave import. At the same time variations are considerable, for in some severe cases the appetite and digestion hold out well throughout, while in others the digestion is the first symptom to fail (this has been called *dyspeptic phthisis*). Other things being equal, the condition of the digestive system is of great prognostic value, for it is often seen that the patient does well so long as the digestive powers are maintained, but fails rapidly so soon as they give way.

Appetite.—The appetite is often good throughout; food is taken freely and enjoyed. More commonly the appetite is capricious, but not more so than in other delicate persons who get little exercise and fresh air. In a few rare cases the appetite is completely lost. Not only is there no taste for food, but an actual repugnance to it, so that it seems almost impossible to swallow it even with the best will to do so. On the other hand, the opposite condition may occur, and the patients exhibit a craving for food which it seems impossible to satisfy. Either of these extremes is rare, but both are of bad omen.

When the digestion fails, the ordinary symptoms of dyspepsia present themselves; in the stomach, pain after food, flatulence, eructations, nausea and vomiting; in the intestines, colic, irregularity of the bowels, and unhealthy motions.

Vomiting.—Vomiting, though by no means uncommon in phthisis, is rarely of serious importance. It occurs as a more or less marked symptom, according to Wilson Fox's figures, in about 30 per cent. of acute cases, and 15 per cent. of chronic, and by preference in the female sex. The causes which excite it fall into two main groups—the dyspeptic and mechanical.

Dyspeptic.—The food taken lies undigested in the stomach, and causes pain and discomfort, as in other forms of dyspepsia, until it is rejected.

Mechanical, as the result of violent coughing. In some cases the mere taking of food excites coughing, which continues until the stomach is evacuated, exactly as occurs in whooping-cough More commonly the paroxysm occurs either the first thing on rising in the morning, or late at night before finally settling down for sleep. In this form, though the contents of the stomach are brought up there is no relation between the vomiting and the taking of food.

Nervous.—These two groups cover together the great majority of instances of vomiting in phthisis, but there remains a third group, in which the vomiting is due neither to the taking of food nor to cough, but occurs without apparent cause. It is often described as nervous or reflex, and is attributed to irritation of the pneumogastric nerve. The irritation is usually referred to two places, either to the terminations of the nerves in the lung, the irritation being produced by the lesions there, or to the trunk of the nerve, the irritation being caused by the pressure of enlarged glands, bronchial or mediastinal.

Cases which are held to prove the latter are recorded chiefly by Guenneau de Mussy, Simmonneau, and others, but the proof is difficult, and the records leave much to be desired.

The other theory which attributes the vomiting to irritation of the peripheral nerve-endings in the lungs is still more a matter of opinion and difficult of proof. The cases usually referred to this cause are those in which the vomiting has been a prominent symptom before the detection of definite physical signs in the lungs. It is a clinical fact worthy of note that in these cases the vomiting usually ceases or becomes less frequent when the mischief in the lungs becomes more pronounced.

In either case it is stated that the vomiting is more likely to occur when the left nerve is involved rather than the right. Legroux asserts this for enlarged glands, and Habershon [1] quotes statistics in support of it for the lungs.

The tongue presents no characteristic change. When there is much constitutional disturbance it is usually coated with a moist creamy fur, which may peel off in places, leaving the surface beneath raw and red. It is rarely dry, unless the patients sleep with the mouth open, or there be some acute complication like pneumonia.

[1] Habershon, *St. Barthol. Hosp. Rep.*, vol. xxiv. p. 135.

Thrush is not uncommon in bad cases and in the late stages, especially when the diet consists chiefly of milk. White patches are then seen on the tongue, gums, fauces, and cheeks, and when removed leave the parts beneath raw and tender. Thrush is of no special clinical significance except as an indication of lowered vitality, and it is easily cured by frequent washing, and the use of glycerine and borax. The tongue may remain clean throughout even in acute cases, as sometimes happens in acute pneumonia. More often in chronic cases it is clean, though raw and red and even beefy, like the tongue of diabetics. At other times it is pale and flabby, as in other forms of anæmia and debility.

Thirst is rare unless there be much fever, or a special complication such as diabetes.

Salivation has been described, but it is rare, and is in most cases due to some local irritation in the mouth, of which stomatitis is the commonest.

A narrow **red line** is often seen at the edge of the gums round the teeth. It was once thought to be peculiar to phthisis, but it is not so, for it is often absent in phthisis and is not rare in other states. It is associated in most cases with anæmia.

The Bowels may remain undisturbed throughout, and that even in acute cases. As a rule there are signs of intestinal disturbance, viz., flatulence, distension, pain, and irregularity of action, diarrhœa and constipation alternating, or the one or other predominating.

Usually the intestinal disturbances vary with the other constitutional symptoms, but they may be among the very earliest symptoms, or predominate throughout over the rest. To such cases the name *intestinal phthisis* has been given, an undesirable term as suggesting confusion with intestinal tuberculosis, whereas all that is meant is that the case is one of pulmonary phthisis with marked intestinal symptoms.

The causes of the intestinal symptoms are (1) the ordinary general causes of intestinal dyspepsia, such as improper or ill-digested food; (2) irritation set up by decomposition of the sputum swallowed; and (3) tubercular lesions in the bowel.

After the lungs, the intestines are known to be the most frequent seat of tubercular lesions. They are almost invariably secondary to the lung changes, and due, no doubt, to infection from the sputum which has been swallowed. Yet there is no necessary relation between the symptoms and ulceration, for extensive ulceration may exist without any intestinal symptoms at all, and the symptoms may be severe without any ulceration.

I remember a child with the most extensive tubercular ulceration throughout the whole of the intestines I have ever seen, the existence of which was utterly unsuspected owing to the entire absence of symptoms.

Tubercular ulceration may produce symptoms:

1. By the congestion and inflammation around which lead to diarrhœa;
2. By interference with the proper peristalsis of the intestine as the result
   - (*a*) Of changes in the muscular coat;
   - (*b*) Of cicatricial narrowing of the gut;
   - (*c*) Of local peritoneal adhesions, or diffuse chronic peritonitis and the pain connected with it.

   These conditions lead in most cases to constipation and irregularity of the bowels rather than to diarrhœa.
3. By acute peritonitis and rupture of the bowel; but these conditions belong to the category of complications.

Intestinal pain is not a common symptom, but it may be severe. It is generally due to colic as the result of distension of the bowel by wind or fæces, but it may be due to local peritonitis, or, as is not unfrequent, to both combined. It is often associated with tenderness, especially over the cæcum. When fistula is present as a complication it may, as in other cases, be the source of great distress.

Phthisis cannot, as a rule, be described as a painful disease, but in some rare cases pain may be the prominent symptom throughout, and in almost every part affected.

The following is the most distressing case I have met with:—

The patient was a man of about 40 years of age, who had had slowly progressing phthisis for about three years when it took on rapid action. Then a tubercular ulcer formed at the commissure of the lips on the right side, which caused great discomfort, and was excised, but with no real benefit, for the wound became reinfected. Later still, independent ulceration occurred in the pharynx, which made swallowing very painful. But worst of all was the pain which during the last two months of life developed in the abdomen. This, which was excruciating and almost continuous, was no doubt peritoneal in origin. The suffering was so extreme and so little influenced by remedies that death came as a relief to both patient and attendants. Such suffering is fortunately extremely rare.

Difficulty or Pain on Swallowing depends upon tubercular ulceration of the pharynx, tongue, or parts about the glottis. These are all comparatively rare complications of phthisis, and the amount of pain they excite varies greatly. Much depends upon the part where the

ulcer is placed. Thus if it be at the commissure of the lips the pain is considerable, as the ulcer often cracks from the constant movement of the parts and bleeds. On the tongue, soft palate, cheek, and back of the pharynx, ulceration may be extensive and yet cause but little discomfort. It is where the ulceration is in the epiglottis or epiglottic folds, or around the glottis itself, that the distress is greatest. Then the pain, or the fear of choking on swallowing, or the cough which swallowing evokes, is often so severe that the patient dreads the taking of food. In such cases relief may be sometimes given by painting the throat with cocain just before food is given, but when the glottis is much involved this may be risky, and may lead to the passage of food into the air-passages. Under such circumstances there is nothing to be done but to feed with a tube.

Difficulty in swallowing may also be caused by the pressure of enlarged glands on the œsophagus in the mediastinum. This is a rare event, but I have seen one or two cases in which it appeared to be the only possible explanation.

In one case the swallowing of anything solid was not only difficult, but also caused paroxysms of coughing and dyspnœa, attacks which had been called asthmatic.

Diarrhœa is usually a late symptom, though it may occur very early and be the prominent symptom throughout. It may be easily excited by slight causes, *e.g.*, errors in diet, cold, or drugs, but it stands in no necessary relation to tubercular ulceration. In the later stages the probabilities are in favour of ulceration, but in the early stages on the whole against it; and some authors maintain, as Fagge does, that tubercular ulceration of the bowel is never met with in the adult as the primary disease, antecedent, that is to say, to tubercular disease in the lung.

Some figures quoted by Wilson Fox show that although, as stated, diarrhœa is more frequent in the chronic than in the acute cases of phthisis, yet it is absent in nearly 50 per cent. of all cases, *e.g.*, in 141 cases of acute and chronic phthisis taken together diarrhœa occurred in only 77.

As a very early symptom in phthisis diarrhœa was said by Louis to occur in 12·5 per cent.

When amyloid disease develops it may lead to diarrhœa, but as this complication is not a common one, and is by no means always associated with diarrhœa, this cause may be practically disregarded.

It has been thought that diarrhœa exercised a favourable influence over the rate of progress of the disease in the chest, *i.e.*, that the chest mischief became less active when the diarrhœa was most marked. This, I believe, is not so, but, whether it be so or not, there is no doubt that the exhaustion produced by diarrhœa may be so considerable as to greatly aggravate the gravity of the case during its continuance, and it is, therefore, a good general rule in practice to keep it in check by drugs.

The stools present nothing characteristic. If the motions are small, pus and tubercle bacilli may be occasionally demonstrated, but when they are copious, bacilli would be difficult to find.

A distinction between frequent evacuations and diarrhœa must be drawn. The motions may be frequent and yet small, and the trouble may then be the result of chronic constipation and impaction of fæces, while in other cases it may be due to irritability of the bowel, especially when ulceration exists in the rectum or sigmoid flexure. These possibilities must be borne in mind if the treatment is to be successful.

Hæmorrhage from the bowel is rare unless it be the consequence of piles or fistula, but it may now and then occur with tubercular as with any other ulceration.

Urinary System.—There are no characteristic changes in the urine in phthisis.

In febrile conditions the urine may present the usual febrile characters, *i.e.*, it may be concentrated, acid, and deposit a sediment of urates. Transient albuminuria may occur, but this is very rare.

In the subacute stages the urine may present any of the characters ordinarily met with in feeble persons, changes which stand in close relation with the digestion. It may be limpid, clear, and light-coloured. It may deposit phosphates or be passed turbid. Phosphaturia is a very common condition in the inhabitants of towns who live sedentary lives and get little exercise. It indicates usually some feebleness of digestion, and is quickly cured by acids and nux vomica. It stands in no close relation with phthisis, though this has been asserted, except so far as it depends upon weak digestion and want of vigour. On the whole, I am inclined to believe it is less common in persons who are actually phthisical than in others.

The urea varies chiefly with the amount of albuminous food taken. Its percentage is roughly normal, *i.e.*, about 1·5 to 2.

The chlorides are said to be normal except when the expectoration is profuse, or pneumonia develops, and then they are reduced.

Albuminuria is rare in the early stages of phthisis, unless it be due to the fever, and then it is transient. In the later stages it is not rare, being then often associated with œdema of the feet, and due, like it, to cardiac weakness. With rest and improved health, both œdema and albuminuria will disappear. If albuminuria be persistent in these chronic cases, it is generally the result of amyloid disease or of granular kidney. Amyloid disease is the direct consequence of phthisis. Granular kidney, on the other hand, stands in no true relation to phthisis at all, and when the two conditions are found together, it is but an instance of the accidental association of two common diseases. Wilson Fox [1] found albuminuria present in 8 per cent. of the acute cases and 33 per cent. of the chronic. Albuminuria has been said to depress the temperature, and it is true that in many cases with chronic albuminuria the temperature is not much raised; but no rule can be laid down, and persistent albuminuria may be found accompanied with a high temperature, 103° or 104°, and in rapidly progressing cases.

Sugar is sometimes found in the urine, either temporarily, when its presence is due to some accidental cause, or permanently. Diabetes, as has been stated, often ends in phthisis, but phthisical patients only very rarely indeed become diabetic.

Ehrlich's reaction is not uncommon in the more active stages of phthisis, but it has no diagnostic value, for it is frequent in typhoid and other fevers.

When tubercular disease of the genito-urinary tract is present, albumen pus, detritus, and tubercle bacilli may be found in the urine. The two diseases are not often associated, and when they are, the genito-urinary tuberculosis has usually preceded that in the lungs.

Nervous System.—There are no nervous symptoms peculiar to phthisis, unless it be the general hopefulness and cheerfulness of the sufferers, which stand in such striking and painful contrast to the prospect, as it appears to others. Sometimes the patients are despondent, gloomy, and even now and then suicidal, but this is rare unless there be a taint of insanity in the family.

Occasionally, before the disease is pronounced, the patients become irritable, emotional, and even hysterical, no doubt from the consciousness of failing health and the anxiety and fear of breaking down. Strange to say, these symptoms often pass away when the patient knows the worst, and they then become resigned and even hopeful.

When hysteria occurs, it is, as in most other instances, the evidence of failing health and the consequence of the disease.

Closely allied to the excitable mental condition is the general cutaneous and muscular hyperæsthesia which is now and then met with in acute cases or during acute exacerbations. The skin over the whole body may then be exquisitely tender and sensitive, so that even light touch and handling may be extremely painful. Usually this is most marked over the chest, and may render physical examination difficult. When local, a little aconite liniment brushed over the painful parts will quickly relieve this and often remove it permanently.

Similarly, the muscular tenderness and aching may be considerable, but, except in the acute cases, it does not, as a rule, last for long. It is generally a toxic symptom, but in some instances may be due to peripheral neuritis, the association of which disease with phthisis is dealt with elsewhere.

In the last stage, as death approaches, the patients may pass into the semi-comatose condition, with general tremors and subsultus, known as the typhoid state, the terminal stage of so many diseases, especially those of a febrile type. It is a toxic state, in which extreme prostration and rapid failure of all the powers rapidly develop.

The other nervous symptoms occasionally met with are the result of some nerve complication, *e.g.*, meningitis, peripheral neuritis, etc.

[1] *Loc. cit.*, 771.

Cutaneous System.—The skin is usually poorly nourished, like the rest of the body. It is thin, semi-transparent, and the veins show beneath it with undue distinctness. It is this, with the absence of fat, which gives the delicate, transparent, bluish, skim-milk-like look to the complexion which is regarded as characteristic of phthisis. The complexion, however, is quite as often thick and muddy, the chief peculiarity in either case being the pallor which develops in the later stages.

Of skin affections, the commonest is *Tinea versicolor*.

This parasitic disease, due to the microsporon furfurans, commences on the front of the chest as small yellowish-brown spots, which coalesce and spread until, in extreme cases, the eruption may extend from the neck to the pubes. Usually it is confined to the front of the chest and to the parts between the shoulders. The fungus is easily demonstrated by scraping a few of the scales off and treating them with caustic potash, when the groups of spores and the mycelium are easily seen. It produces no symptoms unless it may be a little itching. It continues indefinitely if not treated, but is rapidly removed by frequent washing with soap, and the application of some parasiticide, *e.g.*, oleate of mercury ointment, or a lotion of hyposulphite of soda. It has no special relation to phthisis, but occurs with frequency in phthisis, because the chest is kept more carefully wrapped up in flannel and less frequently washed.

Sudamina are not as common as might be expected from the amount of sweating which occurs, and in this respect phthisis stands in contrast with rheumatic fever.

Branny or *furfuraceous desquamation* has been described, but it is not so frequent as in typhoid fever for instance. It is to be associated with the rapid loss of flesh and impaired nutrition of the skin rather than with sweating.

Chloasma, i.e., yellowish-brown pigment stains, are sometimes seen upon the face or other parts of the body in phthisis, but their presence is purely accidental. The occasional association of phthisis with *Addison's Disease* has been already considered.

Herpes has been described, but its occurrence is either accidental or due to the onset of pneumonia.

Purpura has been also met with now and then, but its occurrence is rare. I have once seen a hæmophilic state develop, and the patient died as the result of uncontrollable bleeding from the gums.

The Blood.—The most characteristic change in the general nutrition is the anæmia, which may be a very early symptom, and which even, if delayed, always becomes pronounced as the case progresses.

This is shown in the blood by a diminution in the red cells and in their amount of hæmoglobin, and this diminution increases with the increase of the anæmia, until in some cases the number of red cells may fall as low as 2,000,000 per cm. In early stages there may be no leucocytosis, but in the more advanced stages there may be an increase up to 15,000 or 20,000. This is probably due to the hectic fever.

When leucocytosis occurs, the multinucleated forms are increased, while the eosinophile cells are diminished.

Since in acute miliary tuberculosis, tubercular pleurisy, as also in tubercular peritonitis and meningitis, there is no increase in the number of white cells, but, on the contrary, often a diminution, it is held by some[1] that leucocytosis is not a part of tubercular disease *per se*, but indicates some additional infection, and is, therefore, not a sign of favourable prognosis.

Generative System.—Of sexual symptoms in men there is little to say, except that in the later stages sexual power and desire are greatly diminished, but only in proportion to the general loss of health. When the disease becomes less acute, and the health somewhat restored, desire and power return.

In women, menstruation, if the constitutional symptoms are at all marked, becomes irregular, scanty, or entirely suppressed, and if phthisis develop about the time of puberty, the catamenia may never become established.

[1] Coles, *Dis. of Blood*, p. 233.

The catamenia may, however, continue with their usual regularity into quite the later stages of the disease, and may even become more copious or profuse. When this occurs it is not a good sign, for the loss of blood increases the debility and anæmia.

As a general rule, the catamenia stand in direct relation with the constitutional condition, being entirely absent where the general health is lost, and returning when it is regained.

The relation between suppression of the catamenia and hæmoptysis has been already considered, and it has been shown that vicarious menstruation has at the most a very doubtful existence.

Conception is of common occurrence even in the later stages of phthisis, and that, too, when the catamenia are irregular or even absent.

Pregnancy runs its natural course, and parturition is normal.

It is after parturition that the breakdown of the mother's health is rapid, especially if she attempt to suckle the child. The risk to the child of being suckled by a phthisical mother has been already pointed out. All things considered, the conclusion seems obvious that it would be best for a phthisical woman not to marry, and that if she marry and bear a child, she should, both for her own sake, and that of the child, abstain from suckling it.

The General Nutrition.—Wasting is a part of the disease, and as such has been already described. The wasting affects all the tissues, although it is said to avoid the liver and bones. The liver certainly is often larger than normal from the deposit of fat. The bones, however, are generally slender, and appear to waste, and it is certain that phthisical patients often lose height, though this is probably due to the wasting of the intervertebral discs rather than of the vertebræ.

The Hair.—The nutrition of the hair suffers, so that it loses its glossiness, becomes harsh and brittle, and falls out or becomes gray.

The falling out of the hair is similar to that met with in other constitutional diseases, such as syphilis or typhoid fever, and, as in those affections, involves the whole scalp. It is a general thinning of the hair, so that the alopecia which results is never partial or patchy, and is rarely complete. It occurs, as a rule, only in the acute cases, and, as was known to Hippocrates, is a sign of bad omen.

Premature grayness of the hair of the head and face occurs also, but is not common in phthisis nor peculiar to it. When not merely accidental, it is to be referred to the general loss of nutrition and failure of health.

The hand.—In most cases of phthisis, as the result of the wasting, the hand becomes thin and the fingers look tapering and slender.

The nails also undergo changes.

1. The natural curves are exaggerated, both from side to side and longitudinally, so that they become incurved towards the palm.
2. The nail also becomes longitudinally ridged or furrowed. Both these changes are referred to the wasting of the matrix.
3. Sometimes transverse ridges develop which correspond with exacerbations of the disease, and are similar to those seen now and then after acute illness of any kind, *e.g.*, a fever.

Clubbing of the Fingers.

This remarkable deformity of the fingers, though known to the ancients, was forgotten for centuries, until attention was drawn anew to it in the beginning of the present century. It was then described as "the Hippocratic deformity of the fingers," under which title Trousseau [1] wrote a good description of it.

It was of old regarded as a primitive sign of phthisis—*Tabidis ungues contrahuntur*; *Tabidis ungues adunci*.

[1] *Clin. Lect.*, iii. 305.

In this affection the terminal phalanges of the fingers become bulbous or club-shaped, being in early cases round like a club, but in extreme cases being flattened, and spreading laterally, so as to be shaped like a spatula. The parts affected are the finger-pads and the tissues around the nail, and the resulting shape is not unlike that met with in cases of chronic onychia.

The change, whatever it is, affects the superficial structures only, for the bones and joints are not involved, while the skin itself is not altered, nor the nail, except that it becomes more curved both from side to side and in its length. The colour is sometimes dusky or livid, but only when there is cyanosis, otherwise the appearance is the same as that of the skin elsewhere.

The minute or microscopical changes are not made out. It is certainly not an œdema, and the consistency of the swollen parts is the same as that of a healthy finger. Buhl[1] states that it is a fibrous thickening of the rete mucosum. Possibly in the chronic cases this may be so, but it can hardly be so in the acute or recent cases.

Fig. 135.
Photograph of clubbed fingers.

The affection is almost always bilateral and symmetrical, and in most cases affects all the fingers alike.

Trousseau states that it develops in the thumb and index finger of the right hand first, and then in the thumb and index finger of the left hand, after which it involves the other fingers in order of size, the little finger sometimes escaping altogether. In the few cases in which I have had the opportunity myself of watching it develop, it has affected all the fingers at the same time, and in about the same degree, but in extreme cases it is in the thumb and index that the changes are most marked.

As a rule it is of slow and insidious development, so that the attention of the doctor is rarely called to it by the patient or the attendants. It may, however, develop rapidly, and, as Trousseau states, it may then be attended with lividity and pain.

In extreme cases it may affect the toes as well, and in the same way, but where hands and feet are both affected, it is usually most developed in the hands.

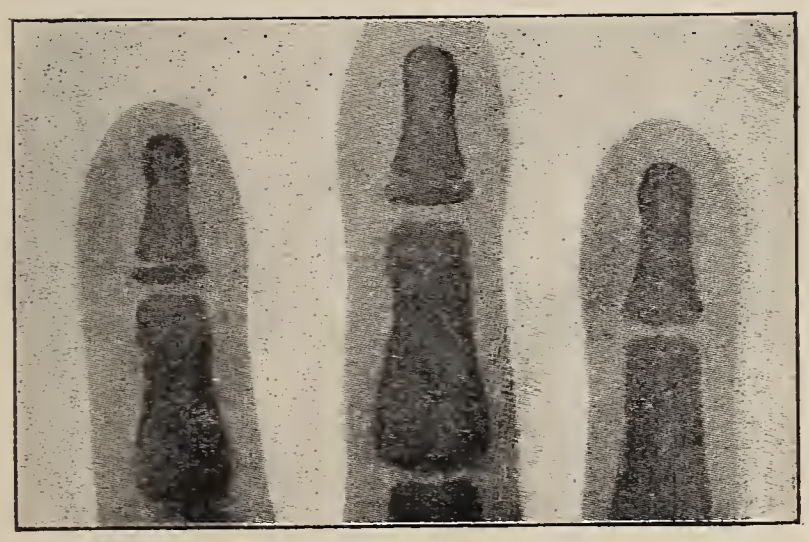


Fig. 136.
Portion of a skiagram (from an illustration by Dr. Frederick Taylor) showing the bulbous condition of the fingers, and the absence of bone changes in the phalanges.

It is chiefly found in cases of chronic phthisis, of chronic empyema or pleurisy, and of congenital morbus cordis.

In *phthisis* it does not occur in more than one-third of all cases (according to Pollock in 29 per cent. of males and 23 per cent. of females), and then in connection with chronic cavities.

Trousseau stated that he had observed it also in connection with *bronchitis* and *emphysema* and with *asthma*, but it certainly is relatively very rare in these affections. I have never seen an instance of it in either. I have notes of one case in which there was no direct evidence of any other disease than emphysema, but as the patient had spat blood several times, I thought it to be chronic phthisis associated with emphysema.

[1] *Lungen-entzündung, etc.*, 1872, p. 159.

It has also been met with in *abscess of the lung*, and in *thoracic aneurysm*, and Smith has described its occurrence in acute *pneumonia*, but this is, so far as I know, a unique case.

But it has also been recorded in cases where there has been no intra-thoracic disease at all, for example, with general *amyloid disease* consequent on chronic suppurative disease of the hip joint,[1] with *cirrhosis of the liver*,[2] and even with a general disease like *purpura*.[3] In all these cases it was bilateral and symmetrical.

Fig. 137.

Skiagram of the foot from a case of clubbing of the toes and fingers in a child with congenital morbus cordis.

There is a still more remarkable series of cases in which it was *unilateral*. Such instances have been described in connection with subclavian aneurysm by Ogle,[4] Canton, and Thomas Smith,[5] the clubbing occurring only on the side on which the aneurysm was seated. Gay has also recorded a case in which there were two subclavian aneurysms, and the clubbing was bilateral. It diminished when the limbs were raised, and disappeared when the aneurysms were cured. In other cases, too, the cure of the aneurysm has been followed by the disappearance of the clubbing.

In the last cases the occurrence of clubbing was naturally referred to obstruction of the circulation through the affected limb, and that explanation has been also given of most of those cases in which the affection was bilateral and cyanosis present, as, for instance, in congenital morbus cordis. But such a mechanical theory is inadequate, for in the first place clubbing is not œdema; next, it is hardly ever met with in association with œdema; or in cases where cyanosis and central obstruction are considerable, as with heart diseases other than congenital, with intrathoracic tumours, with bronchitis and emphysema, or with asthma; finally, it occurs in many thoracic cases where there is no obvious interference with the circulation at all, as in chronic empyemata; and occasionally also in abdominal conditions in which the thorax is entirely free from disease.

The cause of the affection is therefore unknown, nor does it help us much to call it a neurotrophic disturbance.

The onset is usually, as stated, insidious and slow, so as to be unnoticed by the patient; but it is sometimes rapid.

Thus Wunderlich records a case in which it developed in five weeks, and I have myself seen it become well marked in less than a fortnight.

The condition is in most cases persistent, probably because it is associated with permanent and incurable disease; but where the disease is curable it may spontaneously disappear, as mysteriously as it came.

Thus Thomas Smith refers to a case of pneumonia, in which it developed rapidly, and disappeared almost as rapidly during convalescence; Church observed the same thing in a case of chronic abscess of the lung. In my own case, already referred to, in which clubbing became well marked in a fortnight, the patient was suffering from an empyema. After the side had been opened and drained, recovery was rapid and complete, and the clubbing entirely disappeared before the end of three months.

A similar case is recorded,[6] in which the clubbing developed rapidly after an empyema had been operated on. It got well rapidly as soon as the empyema was cured.

Mangelsdorf[7] records a very striking and interesting case in connection with purpura, where, after a few weeks' illness, both wrist-joints became swollen and painful, and soon after the fingers

[1] Thomas Smith, *Path. Soc. Trans.*, xxiii. 78.
[2] Flüchiger, *Wien. med. Wochensch.*, 1884, 1458.
[3] Mangelsdorf, *ibid.*, 1885, 362.
[4] *Path. Soc. Trans.*, xvi. 428.
[5] *Ibid.*, x. 103.
[6] *Ugeskrift. f. Laeger*, 1895, No. 6.
[7] *Wien med. Woch.*, 1885, p. 362.

began to get clubbed on both sides. In three weeks' time the clubbing was well marked. Its development was attended with lividity of the finger-tips, with pains and loss of sensation. The patient ultimately recovered, aud three months later all signs of clubbing had disappeared.

I have seen two cases in perfectly *healthy persons.*

The first case occurred in a woman of 50 years of age, who came under observation because she had taken some ammonia by mistake, and suffered from troublesome gastritis in consequence. The clubbing[1] had been noticed by the patient herself before the accident, and while she was, as she thought, in perfectly good health. Her attention was drawn to it by the fact that she could not wear her usual gloves and had to get larger ones. Both fingers and toes were equally affected, and at the time the patient was seen the clubbing had only existed for three or four weeks. She passed away from observation, and I cannot say whether the condition was permanent or not.

The second case was that of a medical man about 38 years of age. The clubbing was well marked in all the toes, in all the fingers of the right hand, and in the thumb and index and middle finger of the left hand, the other two fingers being only slightly affected. The clubbing was first observed about the age of 6, and came on without obvious cause. At about the age of 30 it became suddenly more pronounced during residence abroad, again without any obvious cause. I examined him carefully, and found him to be in perfect health, as he stated he was and always had been.

In a third case, a man of 35, with extreme clubbing of both fingers and toes, no cause could be found. He had had pleurisy on the right side, five years previously, which had left no traces whatever behind it.

Myoidema.

Myoidema is a local contraction of the muscle, produced by direct percussion, and causing a nodular swelling, which rises immediately after percussion, lasts a second or two, and then gradually subsides. A second or third percussion in the same spot will reproduce it, but with less intensity each time, until the power of eliciting it is for the time lost. In the pectoralis major it is most marked, usually where the muscle is thin, close to its origin near the sternum, but it occurs also commonly in the fleshier parts of the muscle. It may be observed in the deltoids and scapular muscles, and occasionally in the muscles of the back.

Myoidema, or, as it has been called, *nodular* contraction, has to be distinguished from another kind of muscular contraction, also evoked under some conditions by percussion, viz., the *fascicular*, that in which a deep furrow is produced along the whole length of the muscle by the contraction of the fasciculus, which corresponds with the point percussed.

The two kinds of contraction, the nodular and the fascicular, are not necessarily, nor as a rule, associated together.

Occasionally a wave contraction is excited by the blow, which, starting from the point struck, passes slowly in both directions towards the origin and towards the insertion of the muscle. Its rate is slow, but it seems to vary considerably in different cases.

Of those further facts which are asserted, that the muscles show no abnormality on microscopical examination, and that they respond readily to direct faradisation, but not to stimulation through the nerves, I have no personal experience.

My own observations, which were confined to myoidema of the pectoralis major, were made upon 246 cases, taken at random in the wards of the hospital, 166 surgical and 80 medical, to which I have added 30 more from other sources. Myoidema was found on the whole in 1 case out of every 3, but it was twice as common in the medical as in the surgical wards, occurring in 1 case out of every 2 in the former, and in 1 out of every 4 in the latter.

Among the surgical cases it was present in the greatest variety of diseases. Some were in no more abnormal condition than the enforced rest and confinement of a broken leg entailed. Out of the 38 cases in which it occurred, 21 were suffering from diseases of which the only common factor was suppuration and its attendant effects upon the constitution (*e.g.*, diseases of the hip or spine, necrosis, compound fractures, amputation, etc.), and 13 from other surgical diseases unattended by suppuration, in which group would be included those already referred to, who were recovering from simple fracture. The other 4 were, in addition to their surgical affection, also phthisical.

Out of the 80 medical cases, myoidema was present in 45, and again in a great variety of affections. Of nerve diseases it was found in hemiplegia, paraplegia, cerebral abscess, locomotor ataxy, and muscular atrophy. It was observed in morbus cordis, both aortic and mitral, in cirrhosis of the liver and kidney, in nephritis, in diabetes, in jaundice, in rheumatic fever, in gonorrhœal rheumatism, in typhoid fever, and, lastly, among diseases of the lung, in pleurisy, acute pneumonia, pneumothorax, empyema, and in phthisis. In 1 case of pleurisy, in 1 of

[1] *Clin. Soc. Trans.*, 1897, p. 60.

pneumothorax, and in 1 of empyema, it was found only on the unaffected side, and in a second case of empyema was much more marked on this than on the side diseased. It is worthy of note that in typhoid it seems to be almost constant, though in the typhoid of children it is usually absent.

The chief interest of myoidema centres in its reputed *relation to phthisis.*

Of the 38 surgical cases in which it was present, 4 only were phthisical, and of the 45 medical cases, only 13 had any affection of the chest at all, and of these 4 were certainly not phthisical. From this it is perfectly clear that myoidema is not pathognomonic of phthisis; yet it certainly does occur very commonly in phthisis, though I have no statistics to show its relative frequency.

I have records of 30 cases of phthisis in my series of 275 in which this symptom was noted. Out of these 30 it was entirely absent in 6, though 5 of these were cases of acute and rapid softening, with most marked constitutional symptoms, so that myoidema is not even a constant symptom of phthisis.

Of the remaining 24, 14 had only one apex involved. In 8 of these myoidema was present on both sides, in the other 6 only on one, while out of these it was present in 2 cases on the side opposite to the lesion. Hence myoidema cannot be a measure of the amount of softening which is going on.

In 10 cases both apices were involved, and in all of these it occurred on both sides, though in 5 of them it was most marked on the side most affected.

With these exceptions, so far as my observations have extended, myoidema is bilateral and symmetrical.

It has been asserted that myoidema is one of the most certain signs of phthisis; that it is an absolutely certain indication of softening deposits, so that in exact proportion to its intensity is the amount of rapidity of lung destruction, and the consequent gravity of the case; that increase of softening, increase of muscular irritability, and loss of weight always go together; and that it is possible by this sign to diagnose between typhoid fever and acute bronchitis on the one hand, and softening tubercle on the other.

To these statements I would oppose the results of my own observations, which show, first, that myoidema occurs in many other diseases besides diseases of the chest, and in many diseases of the chest besides phthisis; that even in phthisis it is not constant, though usually present, and that when present it cannot be accepted as a certain index of the amount of tissue destruction, for it not uncommonly occurs on both sides, when only one apex is affected, or it may be absent on the affected and present only on the non-affected side.

Dr. Stokes, who first described this phenomenon, writes thus: "There is nothing in this muscular irritability peculiar to phthisis, but that it is commonly connected with irritation of the lung or pleura there can be no doubt, and in this way, like other signs of irritation, it becomes available in the diagnosis of phthisis. It is always most evident in the earlier periods; and in incipient phthisis occurs over the primary seat of irritation, while in confirmed and chronic cases we may often find it absent over the lung first diseased, and strikingly marked over the side last and least engaged." These conclusions my own observations confirm; and I would differ only in attaching to myoidema still less clinical value in the diagnosis of phthisis.

Myoidema being a symptom common to so many diseases, it is difficult to see with what condition it is essentially associated. Unless it be a reflex act, which is improbable, it must depend upon a direct stimulation either of the muscle itself or of the nerve-endings in the muscle.

Thinness, or the absence of subcutaneous fat, is one important factor in its production; and the explanation of this is simple. A thick panniculus adiposus would render the blow diffuse; when it is thin the blow reaches the muscle in sufficient intensity to set up contraction. But thinness is not the only factor, or it would be constant in all thin people; and this is not the case. Something more, then, is necessary, and that something more must be sought in the over-excitability of the muscle or its nerve. I have seen myoidema, as a part of general nervous exhaustion from over-work, disappearing after rest and a holiday. In some people it is probably physiological, in the same way that the *tâche cerebrale*, though commonly occurring in meningeal irritation, is by no means always indicative of that disease. It is, however, far more commonly associated with some nutritive disturbance in the muscle itself. I have observed it in a muscle which was ill-nourished for want of sufficient use, in which case it disappeared after free exercise. It is common when the muscle is degenerating. It was more marked than I have ever seen it before in a recent case of muscular atrophy, where it was associated with fibrillary twitching, with fascicular contraction, and with so excessive an electrical contractility that the muscle responded to the weakest current the battery could produce. It is, again, very common, almost constant, in that atrophy of muscle which is part of the general emaciation of hectic or of severe disease; and its frequency in typhoid is suggestive when considered in connection with the degeneration of muscle so common in that fever. Lastly, it may be associated probably

with the exhaustion of a muscle from over-work. In this way it seems reasonable to explain its occurrence in empyema, pneumothorax, and in phthisis on the non-affected side, and its absence on the side diseased.

Myoidema seems, then, to be due, apart from mere thinness, which is so important for its production, to an over-excitability of the muscle or its nerves, and it may be either physiological, or, as is more usual, an evidence of some nutritive disturbance in the muscle itself, and this may depend on local disease in the muscle tissue, or be part of some general impairment of nutrition.

In any case myoidema occurs so frequently, and under such variety of conditions, as to be a sign of little clinical value in diagnosis.

Pulmonary Osteo-arthropathy—Arthropathie Hypertrophique Pneumonique (Marey).

Pulmonary osteo-arthropathy is the name given to a group of cases in which an affection of the joints is associated with chronic chest disease, and, as the term implies, is attributed to them. The chest diseases with which these affections are associated are various: chronic empyema, chronic cavities in the lungs, and chronic phthisis, *i.e.*, diseases attended with prolonged suppuration, but not necessarily tubercular. The osteo-arthropathies are also multiform, but the cases described fall into two groups, according as there is actual disease of the bones or not.

The parts affected are usually the small joints of the hands and feet, and sometimes the larger joints, such as the wrist and ankle, knee and elbow, or (rarely) the hip and shoulder.

I. In the one group the swelling is in or round the joints, and there are no bone lesions, as may be demonstrated by the X-rays. These swellings appear to be always associated with clubbing of the fingers and toes, and, like the clubbing, may disappear when the original disease is cured. They closely resemble the joint swellings met with in gonorrhœal rheumatism. They are probably due to the absorption of toxin.

II. In the second group there is very extensive change in the bones about the joints as well as in the shafts of the bones. The bone lesions are those of chronic periostitis, with much thickening of the bones; the joint lesions those of chronic arthritis with erosion of cartilages. In some instances the lesions are remarkable for their wide diffusion.

Thus in a case recorded by Thorburn,[1] which closely resembles a much earlier one described by Bamberger, all the bones of the extremities, small and large, were similarly affected.

The difficulty in all these cases is to prove the causal connection between the lesions in the bones and those in the chest, and to disprove the possibility of their both being the results of some common cause, *e.g.*, tubercle or syphilis.

Thorburn's case is instructive in this respect, for the man, who died at 25 years of age of amyloid disease, began his illness at the age of 16 with osteitis of the head of the right tibia, followed a little later by spinal caries. At 18 the swellings of the hands and feet appeared, and it was not till a year later, at the age of 19, that chronic mischief was discovered at one apex. At the time of death, besides the bone lesions referred to, old tubercular mischief was found at both apices, in the right tibia, in the spine, and in the supra-renal capsule. It is possible, therefore, that all the lesions were of the same nature, *i.e.*, of tubercular origin.

In such cases it seems to be begging the question to regard all the bone lesions as the result of the changes in the chest, when they may all be only the results of a common cause.

In the first group the disappearance of the swellings when the chest disease is cured renders their dependence on the chest affections reasonable.

In the second group, osteo-arthropathies of the kind described are so rarely associated with chest affections as to fairly raise the question whether, if not both due to some common cause, *e.g.*, tubercle or syphilis, they may not be instances merely of accidental association.

In phthisis, at any rate, osteo-arthropathy is so uncommon that it may be practically disregarded as a complication.

The diagnosis has to be made from rheumatic osteo-arthritis of the ordinary kinds, from osteitis deformans, from chronic tubercular and syphilitic affections, and lastly from acromegalie, and in this connection it is interesting to remember that the earliest case recorded by Marey proved ultimately to be one of acromegalie.

[1] *Path. Soc. Trans.*, xlvii. 177.

The affection was first described and the name invented by Marey in 1891. About the same time, and independently, Bamberger [1] described similar bone lesions in connection not only with chest affections, but also with heart disease. One of the most important recent papers on the subject is that of Godlee.[2]

THE FORMS OF PHTHISIS.

The Pathological Forms.—The prime pathological changes which have been described, viz., nodular and infiltrated tubercle, caseation, excavation, and fibrosis, are combined in very varying proportions in different cases, so that hardly any two cases of phthisis present exactly the same features. Yet the family likeness between them all is so striking that there is full justification for the conclusion drawn by the earlier writers of this century that they were all alike forms of the same disease. It was only when the minute details of the lesions came to be closely studied, that the differences then observed seemed also to justify the classification of the cases into different categories, and, according as the older observers laid more stress upon the general family likeness or upon the variation in the anatomical details, so were they led to uphold the doctrine of the unity or diversity of phthisis. The relation of the nodular tubercle to the caseating infiltration, and of one or both of these to caseous pneumonia, remained a matter of opinion until the discovery of the bacillus. This finally determined the relation existing between the acute tubercular processes and the lesions of phthisis, and settled at the same time the question of the unity of phthisis.

Still the differences which have been referred to justify the classification of tuberculosis of the lung into different forms, which, though the result of the same cause, yet differ widely in their clinical course and history as well as in their pathological anatomy.

Thus acute general tuberculosis stands apart, and is rightly separated from phthisis as ordinarily understood, *i.e.*, from the more localised and destructive forms of tuberculosis of the lung. The forms of phthisis differ most, *inter se*, in respect of the amount of acute inflammatory change, or of fibroid induration, with which they are associated. In the most acute the inflammatory lesions predominate; in the most chronic there is little, if any, active inflammatory mischief, but fibroid changes exist almost alone, or with more or less of excavation. Between the two extremes come many intermediate stages, in which the three prime lesions, inflammation, fibrosis, and excavation, are variously combined, so that the forms of phthisis may be conveniently arranged into three groups, acute, subacute, and chronic, a grouping which fits in as well with the clinical condition as with the pathological lesions. No classification of this kind can be perfectly satisfactory, for on the one hand the different groups merge insensibly into one another, so that no definite line can be drawn between them; and on the other, the same case at different times may belong to different groups, for the most acute case may become chronic, and a chronic case pass again into an acute or active stage. It only tends to confusion to multiply names in order to express supposed differences where none really exist, for the various forms of phthisis differ only in degree, and not in kind, and the very indefiniteness of the terms, acute, subacute, and chronic, are their chief recommendation.

It is very probable that tubercles may even abort and lead to fibrosis without previous caseation. This may occur even in the miliary disseminated form, the "gray granulation of Bayle" being only a miliary granulation which has undergone fibroid change. The same explanation may also probably be given of some of the cases of diffuse fibrosis of the lung, so that the term fibroid phthisis may be rightly applied to them as being of tubercular origin.

[1] *Ztschft. f. klin. Med.*, 1891, p. 193.

[2] *Brit. Med. Jour.*, 1896, July 11 and 18.

The forms of tuberculosis of the lung may therefore be arranged in complete series, commencing with the soft acute granulation, and ending with fibrosis, thus :

1. Acute tuberculosis—
   (*a*) miliary.
   (*b*) nodular.
2. Phthisis—
   (*a*) acutissima, acute pneumonic or inflammatory phthisis (galloping consumption).
   (*b*) acute caseating pneumonia with recent cavities.
   (*c*) subacute, with caseation, excavation and fibrosis.
   (*d*) chronic, with predominance of fibroid changes and excavation.
3. Chronic general induration (some forms of "cirrhosis of the lung" or "fibroid phthisis").
4. The gray granulation of Bayle.

The Clinical Forms.—The clinical varieties of phthisis group themselves most conveniently in the same way as do the pathological, into the acute and subacute, *i.e.*, the more or less actively progressive forms, and the chronic, *i.e.*, the more or less stationary forms.

The acute is characterised by marked constitutional signs and rapidly progressing physical signs; the chronic, by few, if any, constitutional symptoms, and by almost stationary physical signs; while the sub-acute form constitutes the large intermediate group, in which the constitutional symptoms are more or less marked, and the physical signs more or less progressive; to this group most of the ordinary cases of phthisis belong.

In *acute phthisis* the constitutional symptoms are severe. The fever is high and of a marked hectic type, with profuse night sweats. The patient looks very ill and loses flesh and strength rapidly. The localising symptoms are marked; the respirations are rapid, the breathing short, and the complexion dusky; the dyspnœa and cyanosis are usually out of proportion to the amount of the physical signs.

The physical signs are at first often quite indefinite, and may not be more than would be caused by slight bronchitis. Localised bronchitis, however, indicates a local lesion, and when limited to the apex, points with great probability to the presence of tubercle there. By-and-bye the signs of consolidation appear, either in scattered patches, as in broncho-pneumonia, or in masses, as in ordinary pneumonia.

The physical signs rapidly progress, and any difficulty of diagnosis there might be would occur only in the early stages.

In the *subacute form*, the constitutional symptoms vary in degree according to the activity of the disease. The chest symptoms are less urgent, but the physical signs are usually definite, showing gradually-increasing consolidation with breaking down and excavation, while the expectoration is characteristic and contains elastic tissue, bacilli, and often blood.

In the *chronic forms*, the constitutional symptoms may be absent altogether, while the chest symptoms and the physical signs vary with the amount of mischief previously produced. If in such a case one lung only be diseased, and the other have undergone complementary hypertrophy, chest-symptoms may be almost absent.

As a rule phthisis is of the subacute form, *i.e.*, it runs a slowly progressive course, but its progress is rarely regular. It advances *saltatim* by an irregular series of exacerbations, with intervals of comparative quiescence. According to the complete or partial nature of the remissions, phthisis has been spoken of as *intermittent* or *remittent*. When the intermissions are complete, the disease may pass into the stationary form, and remain *in statu quo* for many years, but in most cases, sooner or later the mischief recrudesces, and passes again into a more or less active form.

On the other hand, even the most acute and threatening cases may become quiescent, with partial or even complete remission.

The different clinical classes into which the cases of phthisis are often grouped are purely artificial, and I do not think anything would be gained by discussing them in detail, as if they were well-marked clinical groups. The symptoms, physical signs, and pathological changes are all the same in kind, indicating consolidation, contraction, and excavation, and differ only in degree and rate of development. The physical signs indicate certain pathological changes, from which an inference may be often drawn as to the duration of the disease, *i.e.*, whether it is of recent date or of long standing; but they do not tell us what we most want to know, viz., whether the mischief is actively progressing or not; this we can learn only from the general or constitutional signs. If the constitutional signs are marked, *i.e.*, if there be rapid loss of flesh and strength, with much fever and sleep sweats, the disease is in an active state, and that whether the physical signs be slight or extreme, or whether they show the disease to be of recent origin or of old date. The case must then be placed in the class of acute or subacute phthisis. If, in the course of time, the constitutional disturbance subsides and disappears, though the physical signs remain the same, the case will pass out of the acute or subacute into the chronic group.

On the other hand, if the constitutional signs be absent, no matter what the physical signs may be, nor even if they showed that a large part of the lung had been for ever destroyed, *i.e.*, whatever mischief the disease may have already done, the disease is not, at any rate at the time, progressing, and the case must be placed in the category of chronic or non-active phthisis.

I shall not, therefore, spend much time upon the different groups of phthisis, except so far as these may present special points for consideration, believing that the general account of the signs and symptoms will be sufficient, and that with them it will be easy to place the particular case in its proper group.

Acute Tuberculosis of the Lung.

Acute pulmonary tuberculosis may be a part of general tuberculosis, or be confined to the lung. In the former case the original source of infection may exist in any part of the body; but in the latter it is generally to be found in connection with the lung itself.

It occurs in two forms, which may be called the miliary and the nodular respectively, the difference consisting in the colour and size of the lesions.

In the *miliary form* the lungs are thickly sown with myriads of small granulations of a gray colour, and with but little evidence of caseation in them. They are seated in the interstitial tissue, and the alveoli are, for the most part, unaffected. This form is, as a rule, the result of infection through the blood vessels.

In the *nodular form* the tubercles are of larger size, and all caseous. They are less numerous, and their longer duration on this account has permitted caseation to take place. These tubercles are in great part intra-alveolar, that

is to say, they are nodules of caseous broncho-pneumonia due to the aspiration into the terminal bronchi of caseous substance from the air-tubes.

If the tubercular masses be less numerous still, they may reach a larger size, even that of a cherry or almond, as Laennec pointed out.

In all forms of acute tuberculosis, the lung tissue between the tubercles, as well as the small bronchi, are in a state of acute congestion, and the symptoms which result are so severe that life is in most cases rapidly brought to an end. In some instances the symptoms may be so severe and acute as to be well described by the name Graves indicated for them—*acute tubercular asphyxia.*

The less disseminated the tubercles are—in other words, the more the process tends to become localised—the more advanced the changes met with will be; so that no definite pathological line can be drawn between these less diffuse forms of general tuberculosis and the acute forms of phthisis, in which the tubercular process, being more localised, has time to undergo further changes, and ends in the excavation which has always been regarded as the essential characteristic of phthisis.

The **diagnosis of acute pulmonary tuberculosis** often presents great difficulties, especially in the early stages.

A. **In acute general tuberculosis** the symptoms at first are those of general septic infection, and the diagnosis of septicæmia or typhoid fever is often made.

The tubercles are of small size, miliary, as they are called, and it is not until the stress of the disease falls on some definite organ, like the meninges or the lung, that the diagnosis becomes clear.

In that form of general tuberculosis in which the disease is of a much more chronic character, the tubercles reach a larger size, so that the cases would be more correctly described as chronic nodular, rather than as acute miliary, tuberculosis.

There may be, then, no other symptom than that of profound, gradually advancing cachexia, and though the lungs may be involved, localising signs in them may be almost absent.

I have seen a case of this kind in a young man of 18 years of age, in whom tubercles existed in large numbers in every organ of the body. They were fairly uniform in size, about half an inch or thereabouts in diameter; they existed in the lungs, spleen, and kidneys, and there were, besides, no less than sixteen tumours in the brain. Besides the profound cachexia, optic neuritis was the only other evidence of disease.

B. When **acute tuberculosis is confined to the lungs**, or affects the lungs chiefly, the symptoms are out of all proportion to the physical signs, *i.e.*, there is more dyspnœa and cyanosis than the physical signs are adequate to explain.

Of these cases there are two classes: the first where general pulmonary tuberculosis develops in the course of manifest phthisis; the second where the patient, at the time, is not known to be tubercular.

If, in the course of a case of phthisis, a sudden aggravation in the symptoms take place, and it be associated in the parts of the lung which have hitherto been free with a development of physical signs such as those of congestion, bronchitis, or pleurisy, even without any actual signs of consolidation, the diagnosis of disseminated tuberculosis is fairly certain.

As a rule the tuberculosis is of the broncho-pneumonic type, and depends upon the aspiration of infective material into the air-tubes.

Where the patient is not known to be phthisical or tubercular, the diagnosis is of greater difficulty.

If moderate fever set in, the diagnosis is probably made of *typhoid fever.*

If more acute symptoms develop and the fever be higher, especially if it be associated with physical signs in the lung, the diagnosis is made of acute *pneumonia.*

In other cases the symptoms are those of *acute pleurisy.*

The two last groups of cases may be spoken of as the pneumonic and pleuritic forms respectively.

The pneumonic form.—There is generally something in the course of the case which shows that it is out of the ordinary; either the fever is of a hectic character, or it is associated with sweating, or it does not terminate by crisis, or physical signs develop in different parts of the chest of a more broncho-pneumonic or disseminated form.

The most difficult cases of this class for diagnosis are the forms which occur in the course of influenza. In these cases the prolonged fever, the disseminated patches of pneumonia, and occasional occurrence of hæmoptysis, may make the diagnosis very difficult.

Discovery of tubercle bacilli in the expectoration would, of course, settle the diagnosis, but there may be no expectoration; or if any, it may be bronchitic, and not contain tubercle bacilli even when the pneumonia is tubercular.

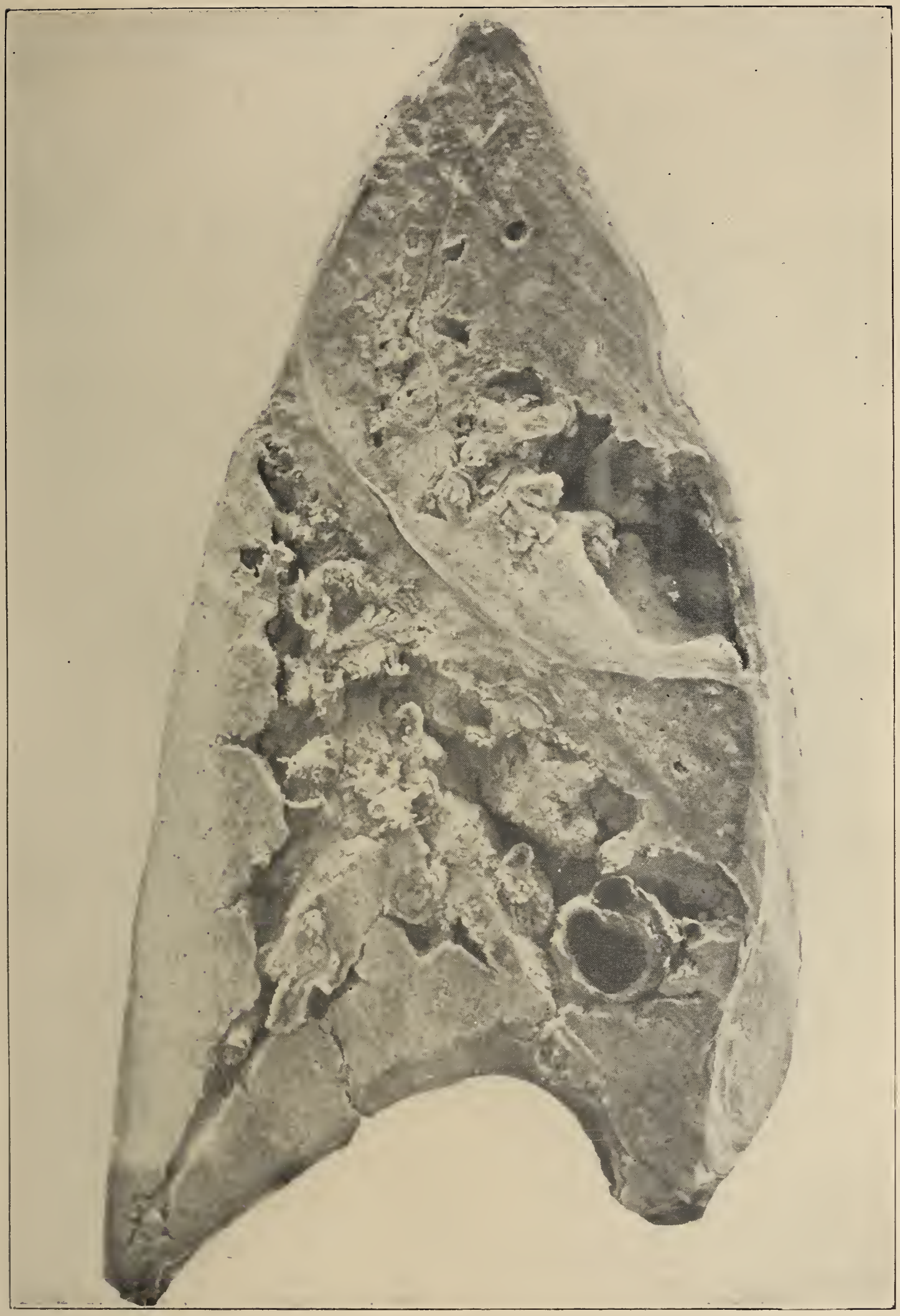
The following case illustrates some of the difficulties which arise in practice.

A schoolboy, 15 years of age, healthy, though coming of a tubercular stock, was seized with feverish symptoms and a little cough, and it was supposed that he had influenza. He got a little better in a few days and was sent home, but the fever continuing, the diagnosis was then made of typhoid fever, and the patient treated accordingly. Shortly afterwards acute symptoms developed in the lung, and the diagnosis was made of acute pneumonia, but before long the case proved to be one of tuberculosis, the very acute symptoms gradually subsided, and the patient passed into the condition of subacute phthisis, and lived for a few months.

This case brought much reproach upon the various doctors at the time, but the difficulties of diagnosis in the early stages were very considerable.

The pleuritic form.—In the pleuritic form there is generally something in the character of the pleurisy which raises grave suspicions of the real nature of the case; either the pleurisy is more pronounced at the apex, or it is widely disseminated over the lung, or it occurs on both sides of the chest. A dry pleurisy widely disseminated, whether it occur on one side or on both, is most suspicious. Often there is an amount of general illness, or of shortness of breath and duskiness of complexion, which is out of proportion with the physical signs.

A man of 30 came under my care with a history of having been poorly for about ten days with pain in his side, and on examination presented the signs of dry pleurisy at the right base. A few days later a small amount of fluid appeared to have developed, but friction was heard much higher up over the side. He looked very unwell and was a little dusky. I felt very uncomfortable about him, and admitted him into the hospital; the temperature was of hectic character and the respirations accelerated. In the course of a week the pleuritic signs had spread over the whole of the right lung, and shortly afterwards they appeared and spread in a similar way on the left; at the same time the signs of effusion at the right base, where the mischief first began, cleared up. All the time the patient was visibly getting worse; the temperature was of a low hectic type, night sweating made its appearance, the patient developed some shortness of breath and cyanosis, and rapidly lost ground. There was no cough for some time, and no expectoration throughout. It was quite clear that the case was one of acute tuberculosis; and the patient died about six weeks from the time of having been, as far as he knew, in perfect health. *Post-mortem* the lungs were found stuffed with recent tubercles, and small miliary tubercles were present in small numbers in almost every organ in the body.



Complete section of the lung from a case of acute phthisis. The greater part of the whole lung is in a condition of acute caseating pneumonia, which is rapidly breaking down in the central portions, especially in the base of the upper lobe and the middle of the lower lobe. Besides these acute lesions, chronic lesions of date long antecedent are evident. The apex is fibrotic, the pleura much thickened, the surface of the lung puckered, and a small chronic cavity occupies the tip of the lobe. A second chronic cavity with thick fibrous walls is seen in the lowest part of the upper lobe, and a third in the lowest part of the lower lobe. The last is nearly filled by a rounded mass with thick walls, and with almost black contents. This is an aneurysm of the pulmonary artery as large as a walnut, completely filled with recently coagulated blood. The section records the clinical history of the case, viz., chronic tubercular lesions of long standing; the large aneurysm shows that the patient had for some time been in imminent danger of profuse hæmoptysis, yet the cavity was so deeply placed as probably to have been undiagnosible; for some reason the tubercular process had recrudesced and developed in its most acute form.

1. The Subacute or Common Form of Phthisis.

When a patient dies of phthisis of two or three years' duration, the condition of the lung commonly found is as follows.

In the apex of one or both lobes are cavities of irregular shape and varying size, the walls of which are formed of pigmented, slate-coloured, fibrous tissue, having either a smooth inner surface or lined with disintegrating caseous substance. The pleural cavity is obliterated over a great part of the lung, and the pleura itself is greatly thickened, especially over the upper parts.

The fibroid induration becomes less as the lower parts of the upper lobe are reached, and patches of caseating consolidation become more numerous, while throughout the rest of the lung, but diminishing in number and size as the base is approached, are more or less abundant nodules and granules of recent tubercle.

The affection begins, as a rule to which there are few exceptions, at the apex, and thence spreads gradually downwards, so that the oldest lesions are found in the upper parts of the lung, and the most recent in the lower parts. Thus every stage and degree of development may be traced in the same case from the gray and caseous tubercle below, through the more extensive caseous patches with recent excavation in the middle, to the fibroid induration and chronic trabeculated cavities of the upper lobe.

2. Acute Phthisis—Acute Pneumonic Phthisis—Phthisis Florida —Galloping Consumption.

In this form caseous pneumonia predominates over the other lesions. The whole upper lobe may be converted into a caseous mass, and the rest of the lungs be studded with caseating broncho-pneumonic patches of varying sizes and with small tubercles.

The cavities are, as a rule, small but numerous, and lined with a thick zone of disintegrating caseous tissue. Though tubercular infiltration is extensive, and tubercles both gray and yellow widely disseminated, there is little, if any, fibrosis, even in the walls of the cavities, which are little else than spots of softening in the midst of recent caseation.

It sometimes happens that there is little or no breaking down at all, so that the whole of one lung or large parts of both are found simply in the condition of recent caseation. This is, however, an extremely rare form of phthisis. I have only met with it twice in a long series of *post-mortem* examinations, and have only seen one or two other cases during life in which I believed that state of lungs to exist.

3. Chronic Phthisis.

Chronic phthisis is characterised by the predominance of induration and excavation. In most cases the upper parts of the lungs contain cavities varying in number and size, surrounded and separated from each other by fibrous tissue, the lower parts of the lungs presenting the less advanced lesions of tuberculosis.

The connective tissue is abundant, dense, and of a slaty colour. The duration of a case of phthisis may be roughly measured by the amount of fibrous tissue present.

The cavities are usually numerous, of irregular shape, and communicate freely with one another. They may, however, be single, and are then usually pear-shaped or more or less globular. They may be quite small; but are often large, and sometimes reach enormous dimensions.

Even the most chronic tubercular lesions may contain bacilli or their living spores, which, if set free, may lead to fresh disseminated lesions, so that any form of chronic phthisis may be found associated with recent tuberculosis.

Although in most cases of chronic phthisis the excavation and the fibroid induration stand in some relation to one another, still there are two groups in which the one or the other greatly predominates.

To that in which induration is in excess, *i.e.*, is out of proportion to the amount of excavation, the term "fibroid phthisis" has been given; while that in which excavation is predominant has an equal right to a special name, and may be described by the term "chronic excavation of the lung."

(i) **Fibroid Phthisis.**—This is a term of somewhat indefinite meaning, being employed by different writers in different senses. By some it is used to describe those cases of phthisis, that is, of tubercular diseases in which there is a considerable amount of fibroid induration. But fibroid induration is a natural part of the chronic tubercular process, and, therefore, no special name is required for it.

By others it is used simply as another name for fibroid induration of any origin, and not necessarily, therefore, tubercular at all. However useful the term may have been in former times, when there were thought to be many kinds of phthisis, it is inconvenient now that the unity of phthisis is established and the term phthisis practically confined to tubercular destruction of the lung. In this general sense fibroid phthisis is simply a pathological expression giving no indication of the cause of the fibroid induration. Thus it may be a local change consequent on local disease, such as interstitial pneumonia, pleurisy, or broncho-pneumonia, or it may be general, and then follow in the course of some general affection of the lung or bronchi, as in some forms of chronic bronchitis, or in dusty occupations; the latter forming the group of pneumo-konioses or dust diseases. To all of these affections, true tubercular disease or phthisis may be added as an after-complication, and this is what is meant by the terms Miners' phthisis, Grinders' phthisis, Millstone-makers' phthisis, etc.

Besides these there is supposed by some authors to be a third group in which the fibroid disease of the lung is a primary and independent affection, the result of some general or diathetic cause, and it is then compared with the fibroid changes in the liver in cirrhosis, or in the kidney in granular kidney. To avoid confusion with ordinary phthisis, *i.e.*, with tubercular disease of the lung, this group is by some writers designated by the term cirrhosis of the lung. In this sense the term is made to include many of the cases already referred to, in which the disease is the result of broncho-pneumonia, chronic bronchitis, or dust inhalation, the only difference being in the excessive production of fibrous tissue. The term then implies that in the author's opinion there is more fibroid tissue than the disease would ordinarily lead to, and that, no other cause being obvious for it, the excessive production of fibrous tissue must depend upon a general diathesis. Diathesis is a term which has been so often used as a cloak for ignorance that it is deservedly discredited, and, before the existence of such a diathesis can be assumed, strong evidence must be brought in proof of it.

There are two general objections which may be urged against the existence of a special fibroid disease of the lungs; the first that in so many of the cases cited as proof of its existence the changes are local, *i.e.*, limited to one lung or even to parts of one lung, while in the affections of other organs with which it has been compared, viz., cirrhosis of the liver and granular kidney, the changes are general,

and involve the whole and not part of the organs, and in the case of the kidney, both organs alike.

The second, that if a general fibroid diathesis existed we should expect to find fibroid phthisis associated frequently with fibroid changes elsewhere in the body, for example, with cirrhosis of the liver and granular kidney, and though this does occur, its frequency is not greater than could be accounted for by the accidental association of such comparatively common diseases.

Putting aside for the moment these *a priori* objections, it will be best to take the evidence for the existence of fibroid phthisis as it was presented by its most recent advocate, the late Sir Andrew Clark.

According to him there are three groups of the disease—

1. Tuberculo-fibroid.
2. Fibro-tubercular.
3. Pure fibroid disease.

In the two latter groups, tubercle and fibrosis are associated together, and the priority of the one or other must be a matter of opinion. Accordingly, Sir Andrew Clark speaks of the tuberculo-fibroid and the fibro-tubercular form; in the former case the tubercular lesion preceding, and in the latter the fibroid.

Of the first form, *the tuberculo-fibroid*, no discussion is necessary, for it is generally accepted that tubercle may lead to extensive fibroid change, and even those who describe these cases as fibroid phthisis mean only that the fibroid induration is in excess, or in unusual amount.

In the second form, *the fibro-tubercular*, where the fibroid disease is supposed to have been primary, and the tubercle secondary, the most striking instances are those which belong to the group of the pneumo-konioses, and would include the diseases known by the names of Miners', Grinders', and Millstone-makers' phthisis.

In the third form, *the pure fibroid*, where there is a well-developed fibrotic change, not due to any obvious cause, it would still have to be proved that the change was not of a tubercular origin, and this it is difficult to do, for it is well known that widespread fibrotic changes may follow tuberculosis of the lungs, and that fibroid induration is indeed its natural mode of cure. Even miliary tuberculosis may undergo fibrosis, and Bayle's "Granulie" is now regarded as nothing more than fibrosed or fibrotic miliary tuberculosis. The racemose fibroid masses, described as peribronchitic nodules by Virchow, are common in phthisis, and are undoubtedly, in most cases, of tubercular origin.

It may therefore be safely stated that all the pathological changes described as characteristic of fibroid phthisis may be the result of tubercular disease. Even in some of the cases published as instances of pure fibroid disease, caseous masses were present in the fibroid tissue, and it is no longer possible to follow Sir Andrew Clark in his statement that caseation is not conclusive evidence of past tuberculosis.

Certainly tuberculosis will cover most of the cases, and evidence of a convincing character to prove that any other cause can produce the fibroid condition of the lung is so far not forthcoming, so that for all practical purposes the conclusion is unavoidable that fibroid disease cannot be shown to be an independent affection.

If pathological evidence fail to prove the existence of pure fibroid disease of the lung, so, too, do the clinical criteria given fail to shake the conclusions to which pathology points, for there is nothing stated of fibroid disease which could not be equally stated of chronic tubercular disease.

The conclusion, therefore, is inevitable, viz., that fibroid phthisis is nothing but a general term, and does not represent any special form of disease.

(ii) **Chronic Excavation of the Lung.** — One of the most remarkable forms of chronic phthisis is that in which complete excavation of a considerable portion of the lung occurs, involving the whole of a lobe, or, it may be, the whole of one lung.

Nothing is then left of the lung tissue but a cavity with thick walls formed by the thickened pleura, marked here and there by ridges which indicate the interlobular septa. Even the large air-tubes and vessels have disappeared, and the only traces of them left are short stumpy projections at the root of the lung. There is, pathologically, no line to be drawn between the common small chronic cavities and these very large ones. They are in all respects the same except in regard to size, and, as with them, there may be nothing in the general condition of the patient to suggest their presence.

The tubercular nature of both large and small alike has been questioned, but there can be no doubt that they are, with but few exceptions, of tubercular origin. Many show evident traces of this origin in the caseous lining they possess; and even when this is not present, it is fair to ask what other lesion than tuberculosis could, in the great majority of cases, lead to this condition.

There is no necessary relation between the size of the cavities and the physical signs and symptoms, for many of the patients are in good health and capable of work. This is due to the marked hypertrophy of the opposite lung, which often is sufficient to ensure complete compensation.

Such cavities, as would be expected, generally develop gradually and take a long time about it. Thus, in many of the cases a history is obtained of some chest trouble for many years, frequently followed after a time by hæmoptysis, which may be profuse; the health in the meanwhile, except for the hæmoptysis, remaining fair, as in the following cases.

Case 1.—A male, aged 27, gave a history of pleurisy eight years ago, followed by good health except for a chronic cough. Three years later he had an attack of profuse hæmoptysis, which was repeated two or three times subsequently. His health was good except for this until eighteen months before he came under observation; he then became ill and unable to work. The physical signs showed that he had complete excavation of the left upper lobe with complementary hypertrophy of the whole right lung.

Case 2.—A man, aged 32, was in good health until attacked without warning by profuse hæmoptysis 7½ years ago. He lost 2 pints of blood. Recovering from this, he went back to work and continued in good health for five years, then another attack of hæmoptysis set in and lasted on and off for two months, after which time he never seemed to have been the same. The physical signs showed complete excavation of the whole right upper lobe, with complementary hypertrophy of the left lung.

Sometimes there is no history of anything but chronic cough for many years, as in the following case.

Case 3.—A man, aged 41, the whole of whose left upper lobe was excavated, had had no illness during the whole of his life, but had been subject to winter coughs since 8 years of age.

In a few instances there is absolutely no history at all of any illness, or, indeed, of any cough.

Case 4.—The most remarkable instance of this I have met with occurred in the case of a young woman whose whole right upper lobe was excavated, the left lung being in a condition of well-marked complementary hypertrophy. The patient looked the picture of health, was fat and well-developed, and there was nothing whatever to point to the chest as the seat of disease, and the condition of the lung was only discovered on systematic examination.

Though, as a rule, a history of chronic illness is given, there are instances in which a short history only is obtainable, as in two of my cases of six months and eleven months only; but of course it is possible, indeed likely, that the mischief was of much older date, and the history only that of the recent exacerbation.

It is true, of course, that now and then, in acute cases of phthisis, excavation is observed to be very rapid, and if the opposite lung be not affected, complementary emphysema may also develop with great rapidity. These cases are, however, exceptional and rare, for in such acute cases, as a rule, both sides are, or soon become, involved.

The course of these cases is chronic, that is, given the cavity, the patients may continue for a long time, possibly years, in fair or even good health, and may be capable of active work. Sooner or later, however, severe symptoms set in. The commonest of these is hæmoptysis, which is often profuse and may be fatal, the cause being the rupture of an aneurysm of the pulmonary artery within the cavity. This hæmoptysis is sometimes recurrent, but, except for the temporary debility it leads to, may have little or no effect upon the course of the case.

As a rule, in the end, after a longer or shorter time, acute symptoms develop in the opposite lung, and the case then runs the course of ordinary acute phthisis. Of course a patient with such damage to the lung is in greater danger when any complication occurs, such as bronchitis or pneumonia, and the old lesion in the lung, in many of these cases, is the presumable cause of the fatal result.

The cavities may be dry, that is, secrete little or nothing. On the other hand, they may secrete freely or even copiously. In such cases the discharge is purulent, and resembles that of an empyema, from which it is not always easy to diagnose it; nor is the diagnosis rendered easier when, as sometimes occurs, the discharge becomes fœtid.

If the cavity be very large, as, for instance, when it involves the whole lung, the physical signs may strongly resemble those of pneumothorax, with which affection I have more than once seen these cavities confounded, for there may be a somewhat tympanitic note on percussion with succussion and bell sound; but the diagnosis is usually easily made, for the percussion note, though tympanitic in character, is flat and boxy; the side is somewhat contracted, and the displacement of organs is into the affected side and not into the opposite side.

I have notes of 25 cases of this extreme excavation, and in many of them the diagnosis made during life was confirmed by *post-mortem* examination. In this series I have included no instance of excavation of anything less than a whole lobe, and in many of the cases the excavation extended beyond one lobe, while in two the whole lung was involved.

Such extensive excavation seems to occur, with but few exceptions, in men only; at any rate, of my 25 cases only 3 were women. Sturge[1] records 2 cases in women. The affection is not commoner on one side than the other, for in 11 the condition was found on the right side and in 13 on the left, but in the great majority of instances it was the upper lobe that was involved.

In one case the lower lobes on both sides were affected, and the expectoration was profuse, amounting to 30 or 40 ounces daily. This occurred in a girl of 13, and was altogether so exceptional as to require, I think, a different explanation. As no *post-mortem* examination was permitted I have not included it in the series.

The condition is fairly equally distributed between the different age periods from 20 to 45, but it is somewhat more frequent, as would be expected, between 30 and 45.

Ages.	13 to 15	—20	—25	—30	—35	—40	—45	—50
No. of cases, .	3	1	2	3	4	5	4	1

[1] *Mic. Med.*, Feb. 1882.

As complete excavation of the whole lung is very rare, I have made a short note of the only two cases I have met with.

Case 1.—Alfred S., aged 39, was admitted into the Chest Hospital, Victoria Park, on the supposition that he had a localised pneumothorax. On account of the dyspnœa, paracentesis was performed. About 8 ounces of pus and some air were removed, to his temporary relief. The physical signs remained as they were, the side being contracted, slightly tympanitic to percussion, the vocal vibration and vocal resonance not increased, amphoric breathing over the whole side, with the bell sound on percussion. The patient was tapped two or three times, and ultimately a valvular trocar was inserted with the view of giving permanent relief, but the patient died of exhaustion a few days later. *Post-mortem* the right lung was found to be in a condition of complementary emphysema, but with little pathological change in it, except for one or two small nodules over which the pleura was retracted, scattered about the upper lobe, and a very chronic pear-shaped cavity lying in its centre. The left lung was completely excavated, not a trace of lung tissue being left, a few coarse trabeculæ only marking the division between the lobes and the remains of the large vessels. Death in this case occurred from exhaustion.

Case 2.—John H., aged 38, was admitted into the Chest Hospital for cough and dyspnœa. He dated his illness from two years back, when he was attacked by cough, shortness of breath, and pains in the chest. The cough had continued and been worse in the winter, but he had never been entirely laid up. The physical examination showed slight contraction of the left side, this being impaired to percussion; the vocal vibrations were absent in front and at the side, but were felt feebly behind; amphoric resonance was heard at the apex on coughing, and the bell sound obtained here.

When the patient had been in the hospital five weeks, and had considerably improved, he was suddenly seized one day with hæmoptysis, and expectorated about a pint of blood daily for the six days following, and on the seventh he had a fresh and more profuse attack, in which he died suddenly. The *post-mortem* examination showed that the right lung was practically healthy, except for one or two small hard nodules here and there. It was in a condition of complementary emphysema, extending nearly two inches to the left of the middle line of the sternum on to the opposite side. The left lung was represented by one large cavity, a few coarse ridges only remaining over the course of the great vessels at the root of the lung. Upon the largest of one of these was a hemispherical, egg-cup-like swelling, about the size of half a cherry. This was an aneurysm half an inch in diameter. On looking into it, a longitudinal slit, about three-eighths of an inch long, was seen; this was an opening leading directly into one of the main branches of the pulmonary artery. Nearly half of this aneurysm had been torn away by the force of the blood. The walls of the cavity presented the ordinary appearance of a chronic pulmonary cavity. There were no other changes of importance in the other viscera.

These cases of chronic excavation, though, as I believe, almost without exception tubercular in origin, and therefore belonging to the category of chronic phthisis, form an interesting and remarkable clinical group, and therefore deserve, it seems to me, special description when speaking of the forms of phthisis.

Latent Phthisis.

The constitutional symptoms and the physical signs bear as a rule a fairly close relation to one another, but there are exceptions. On the one hand, the constitutional symptoms may be well marked, but the physical signs be so indefinite that the diagnosis may for a long time be in doubt. On the other hand, the physical signs may be well marked, and show the existence of even extreme excavation, and yet the constitutional symptoms be completely absent, the patient being apparently in perfect health and giving no history, at any time, of chest trouble or illness. To both these forms the name of latent phthisis has been given, but it is clear that it does not apply in the same sense to them both.

In the former group the symptoms are present, but they are either not recognised or their meaning misinterpreted.

In the latter there are no symptoms at all, and only physical examination reveals the actual condition of the lungs.

For instance, a young adult who is losing health and strength, and looks delicate and ailing, may be regarded as suffering from anæmia or general debility, until more careful examination shows that there is some elevation of temperature and shortness of breath. Suspicion once aroused, the diagnosis of phthisis is probably ere long arrived at.

Or, again, it may be recognised that the patient is ill and suffering from a more or less marked hectic fever, but this is referred to some other febrile disease, for example, to disease of a joint, suppuration somewhere, or even typhoid fever.

Where some disease of this kind is actually present, *e.g.*, a suppurating joint, it may be a matter of the greatest practical difficulty to determine what the exact condition of the lungs is. It would be better to describe such cases as *doubtful*, *ill-developed*, or *insidious* rather than latent phthisis, for the phthisis does not lie hid, it is only not recognised.

The term *latent phthisis* would then be reserved for the cases in which symptoms are absent.

In this category, even when so defined, there are four distinct groups of cases.

1. That, in which old and unsuspected tubercular lesions are found on *post-mortem* examination, in persons who have been regarded as healthy all their lives and have died of other causes. Reference has already been made to the great frequency with which such lesions are found *post-mortem* in apparently healthy persons.

2. That, in which in apparently healthy persons profuse hæmoptysis occurs, or pneumothorax develops, without apparent cause. These cases are found, almost without exception, to be of tubercular origin.

3. That, which in former times was described as "phthisis ab hæmoptœ," *i.e.*, where profuse unexpected hæmorrhage marked the commencement of acute phthisis.

In most of these the lesions were either so small or deep-seated that they could not yield physical signs sufficient to establish the diagnosis with certainty.

4. There remains one other small but interesting group in which the mischief is so extensive that diagnosis by the physical signs is perfectly easy, and yet there is little and sometimes nothing in the general health or history to indicate that extensive disease is present.

Most of these cases belong to the category of chronic excavation, and examples have been already given under that head.

COMPLICATIONS OF PHTHISIS.

Many of the so-called complications of phthisis hardly deserve the name, for they are no more than exaggerations of the ordinary symptoms of the disease, *e.g.*, hæmoptysis, dry pleurisy, and bronchitis.

Others, again, though closely connected with the disease, are not such common incidents in it, and therefore deserve to be spoken of as complications, *e.g.*, pleuritic effusion and pneumothorax.

Another group is formed by those affections which are the consequence of secondary tuberculosis in other organs, produced either by local extension to neighbouring parts by direct infection, as in the case of the intestines or larynx, or by general dissemination.

To another group belong such affections as anæmia, thrombosis, general dropsy, and amyloid disease, affections not peculiar to phthisis, but the consequence of the cachexia caused by it. Besides all these affections, phthisis

may be associated independently or accidentally with any other disease, *e.g.*, pneumonia, gangrene of the lungs, nephritis, diabetes, etc.

Most of these affections have been already sufficiently dealt with, and require no further reference here. A few, however, remain which deserve further notice, either because of their intrinsic interest or because of the influence they exercise upon the course of the disease, *e.g.*, pleuritic effusion, pneumothorax, gangrene of the lung, laryngitis, tubercular meningitis, tubercular peritonitis, general tuberculosis, tubercular ulceration of the bowel, and fistula.

Pneumothorax is a very grave complication, but fortunately not very common. The statistics as to its frequency vary a good deal from 1 up to 10 per cent.

Among fatal cases of phthisis my own figures yield a percentage of 5; those of Weil give 4; and those of King-Chambers 3·7 per cent. On the other hand, in fully 90 per cent. of all cases of pneumothorax the cause is found to be phthisis.

Pneumothorax in phthisis usually sets in with very severe symptoms, and yields rapidly to death.

Thus of 39 cases 2 died within 1 hour.
" " 10 " 24 hours.
" " 18 " 7 days.
" " 21 " 14 days.
" " 27 " 1 month.

In other words, 54 per cent. died within a fortnight, and 70 per cent. within the month. Weil's figures yield a mortality of 25 per cent. for the first week, and 50 per cent. for the month.

In most cases death is caused by a more or less rapidly increasing suffocation, but in a few instances it occurs at once from shock, and in others more gradually from exhaustion or from the effects of pleuritic effusion.

Generally the pneumothorax leads to effusion, which is purulent or sero-purulent in 2 cases out of 3, but in the third is serous. When serous, the fluid may be absorbed in the usual way, and recovery take place in time; but with purulent effusion life is rarely prolonged.

In a young woman of 23, with acute phthisis, left pneumothorax developed without cause or other symptoms, except a fall of temperature of 6·8 degrees. It was not until the next day that dyspnœa began to develop, and forty-eight hours later it became urgent, so that paracentesis was necessary. Besides the air, a small amount of sero-purulent fluid was obtained. Three days later paracentesis was again performed in the expectation of obtaining more fluid, but only a few ounces were drawn off. A fortnight later a small suppurating spot appeared in the skin in the seat of the puncture. This was thought to be due to a pointing empyema, and two days later it was incised. Immediately the skin was cut the patient died suddenly from shock. The *post-mortem* showed the presence of a pointing empyema; the lung was collapsed and could not be distended owing to adhesions, but the perforation was closed.

It has been generally accepted, that to treat these cases of pyo-pneumothorax by incision is to shorten their life. I do not accept this view. On the contrary, I believe that if these cases were treated by early incision the results would be more favourable than if they were left alone. I am, at any rate, able to quote some cases of success (*cf.* Pyo-pneumothorax).

In a few cases no effusion forms at all; the air is then readily absorbed and recovery is complete. This may occur even in rapid phthisis, and I have seen a patient recover completely from pneumothorax within a fortnight, though she was at the time dying of phthisis, and actually did die of it a week later.

The most remarkable case of this kind in my own experience is the following:—A young man, aged 32, had been under my care with phthisis of about six months' duration. He was admitted into the hospital, and a month later suddenly developed pneumothorax on the left side, for which he required tapping four times within two or three days. The temperature, which had previously been hectic, and had at times reached 104° (the average maximum, however, being 102°), immediately fell on the occurrence of pneumothorax, and from that time rarely rose more than a point or two above the normal. No fluid formed, but the air was not absorbed, and two months later, when the patient left the hospital, the air was still present and the organ displaced to as great an extent as at first. The patient is believed to have continued *in statu quo* for some time longer, and was then lost to observation.

Latent pneumothorax.—The onset of pneumothorax in phthisis is generally marked, as would be expected, by severe and urgent symptoms; but strange as it may seem, there are cases in which it may develop without any symptoms at all, and be discovered, as it were, by accident. This form has been well described by the term *latent pneumothorax.*

I remember a young woman confined to bed with extensive and rapidly-advancing phthisis, who was found one morning to have pneumothorax, which must have developed during the preceding night, and absolutely without any symptoms. It was discovered only accidentally during the ordinary routine examination of the chest, by the displacement of the heart and other organs. Subsequently 80 ounces of pus were removed, but there were throughout no special symptoms at any time which pointed to the existence of pyo-pneumothorax. Influenced by the dominant teaching, I did not venture to have the side opened, as I should now, and the patient soon afterwards died.

Latent pneumothorax is, of course, much less likely to be met with when the phthisis is advanced. Most of the instances of it occur in early, or what may be called latent or unsuspected phthisis, for even when pneumothorax occurs in the apparently healthy, the cause proves generally to be a definite, though unsuspected, tubercular lesion.

The treatment of pneumothorax is fully dealt with elsewhere. All that is necessary to say here is that if the symptoms are urgent, the air must be let out as often as necessary by repeated paracentesis, but it is well to let the lung remain collapsed for a time if possible, in order to give the perforation time to close, as it often readily does. When effusion forms, it must be dealt with on general principles, according to the amount and nature of the fluid. Serum, as stated, is sometimes spontaneously absorbed. Pus, however, will probably require to be removed, and, as I have stated, the objections to free incision are not so great as is commonly believed, unless operation is postponed for so long as to be useless.

In any case where paracentesis is necessary, the aspirator, if employed at all, must be used with the greatest caution. It is a dangerous instrument in these cases, for if much suction is used the perforation may be easily re-opened, or a fresh one made.

Pleuritic effusion, occurring in the course of phthisis, may be serous, purulent, or hæmorrhagic.

Hæmorrhagic effusion is, of course, the most rare; the amount of blood is usually small, so that the effusion is little more than serum slightly stained with blood. If the blood be in any quantity, it is more likely that the cause is cancer rather than tubercle. When an effusion occurs in the course of known phthisis, the presence of a small quantity of blood in it is of little significance. Its importance is rather in those cases where what seems to be a simple pleurisy turns out to be hæmorrhagic, for although in a simple acute pleurisy the fluid may be sometimes blood-stained, in the majority of instances such a pleurisy proves to be of tubercular origin, and, therefore, the presence of blood becomes of diagnostic value.

Serous effusion, if in small amount, exercises but little influence on the course of phthisis, and may spontaneously disappear as it came, and sometimes with remarkable and unexpected rapidity. If in large amount it aggravates the symptoms seriously, and may require speedy removal. Still, even large effusions may run a favourable course and be completely recovered from.

As to the effect of serous effusion upon the course of phthisis opinions have differed widely. Thus Louis stated that its influence was unfavourable. This was also Walshe's opinion, and my own experience leads me to the same conclusion. Flint regards it as without influence altogether, while it has even been supposed to check the progress of the disease in the compressed lung, as well as to check and control hæmorrhage. I believe the theory to be unsound, and I have no doubt, if allowed to influence practice so far as to prevent the removal of a large and persistent serous effusion, that it is mischievous. It is true that sometimes the effusion seems to have no effect on the phthisis at all, or that the patient may even improve for the time, but, on the other hand, I have on many occasions seen phthisis make very active progress after the effusion, and, I have thought, in consequence of it.

Serous effusions in phthisis should be treated on general principles, and the indication for paracentesis are the same as in the non-phthisical cases.

Empyema is always a very grave complication, for it materially shortens life, but fortunately it is not common.

It must be treated on general principles. Though empyemata are sometimes cured by paracentesis only, this is rare in phthisis, unless the empyema be localised, and then there is no strong objection to free incision. If the empyema be a large one, it has long been taught that free incision should be avoided as only likely to accelerate the inevitable end. I do not agree with this, and, as in pneumothorax, I believe the results would be more favourable if operation were not so long deferred.

Gangrene of the lung is rare in phthisis. It is no necessary part of the disease, and is due to accidental contamination with putrefactive organisms. Reference has already been made to the fact that phthisis is very often a mixed disease, and that the lung may become infected with other organisms than those of tubercle, for instance, those of pneumonia or suppuration as well as of gangrene. That a phthisical lung would offer a suitable nidus for such infections, might be *a priori* expected, and that gangrene and phthisis are not more frequently associated is somewhat remarkable.

The symptoms of gangrene are fœtor and fœtid sputum, but these are not sufficient for the diagnosis, as they may be equally due to fœtid decomposition of the contents of cavities. In such cases the diagnosis would turn most probably upon the general condition of the patient, for the mere fœtid decomposition of the contents of cavities is often unattended with any change in the general condition, and the fœtor may be even temporary and transient. With gangrene of the lung the patient passes into a low depressed septic state, and quickly dies of asthenia.

Gangrene in phthisis is rare. In 200 consecutive *post-mortem* examinations in phthisis I did not meet with a single instance of gangrene. Many authors place the percentage below 1. Wilson Fox's much higher percentage, I think, must have been accidental.

Aphonia is not uncommon, especially in women, and is due to the common causes. The patients speak in a whisper; the voice is lost, but is not hoarse as in laryngitis, and it comes and goes at random. The cords are white, but do not approximate on phonation.

Paralysis of the vocal cords, from pressure upon the recurrent laryngeal nerve by enlarged glands within the thorax, has been given as a cause of laryngeal trouble in phthisis. Such conditions are rare, and the paralysis thus produced affects one cord only. This may be unattended with any alteration in the voice, and be diagnosable only by the laryngoscope.

Laryngitis.—Hoarseness is due in most cases to laryngitis. Phthisical patients are subject, like other persons, to the ordinary forms of laryngitis, but if the hoarseness be persistent the presumption is in favour of tubercular disease, and laryngeal examination usually proves the presumption correct.

Tubercular laryngitis occurs in two forms—that of extensive infiltration and of ulceration, and usually the two are combined.

The symptoms are hoarseness, cough, expectoration, pain, difficulty on swallowing, and, if there be much infiltration, stridor and dyspnœa.

The hoarseness may be of every degree up to complete loss of voice.

Cough is always troublesome, and, like the voice, is hoarse. It may be paroxysmal, and attended with pain and soreness referred to the larynx.

Pain is often felt on coughing, and sometimes on swallowing, but only when the parts about the glottis or epiglottis are involved.

Tenderness over the larynx is rarely present, unless there be inflammation about it or the cartilages be involved.

Expectoration is scanty, the chief part coming not from the larynx but from the lungs. It may be blood-stained, but copious hæmorrhage from the larynx, though recorded, is extremely rare.

Difficulty on swallowing is most likely due to ulceration round the glottis or in connection with the arytenoid cartilages or epiglottis. Sometimes the pain on swallowing is so severe that it interferes with the taking of food, and may then require the use of cocain.

Stridor and laryngeal dyspnœa are on the whole rare. They are due to two causes; the one being great infiltration and swelling round the glottis; the other fixation of the cords in the mesial position owing to adhesions in the arytenoid joints.

In spite of the amount of infiltration which occurs in tubercular laryngitis, stenosis is not nearly so common as in syphilis.

When stenosis is present, dyspnœa may become very suddenly urgent, and require immediate tracheotomy. Kidd's [1] statistics yield only 0·1 per cent., *i.e.*, 4 instances in 4000 cases.

The relative frequency of the different symptoms is thus given by Morell Mackenzie:

Hoarseness, ...	67·4 per cent.
Dysphagia, ...	30·3 ,,
Aphonia, ...	24·6 ,,
Stridor, ...	1·6 ,,
Tracheotomy, ...	0·6 ,,

Tubercular disease of the larynx is, almost without exception, found associated with tubercular disease of the lung, and secondary to it. Even in a case of primary laryngeal tuberculosis of the larynx, if ulceration occurred, the lungs could hardly fail to become involved, so that laryngeal phthisis, in the strict sense of the term, can hardly be said to exist, and in the ordinary sense of the term it means no more than that the case of phthisis has well-marked laryngeal symptoms.

[1] *Lancet*, 1888, i. 618.

The *frequency* of laryngeal tuberculosis in phthisis is variously stated. Thus Heinze places the percentage as high as 33, while Lebert does not put it higher than 8. So far as symptoms are concerned, I think Lebert's statement is nearest to the truth, for in many cases of phthisis, signs of tubercle, and even of ulceration in the larynx, are found when there was no evidence of its presence during life.

Though most common at the time of life when phthisis is most prevalent, laryngeal ulceration may occur at any age, and instances are recorded[1] very early in life, *e.g.*, at the ages of 3, 4, 7, and 8 years, and even in infants of 11, 12, and 18 months. Among adults it is more frequent in males than females in the proportion of 3 to 2.

The *effect* of tubercular laryngitis upon the course and duration of phthisis is variously stated by different authorities. Thus Morell Mackenzie seems to say that it has no special effect, and quotes many instances of long duration, and even some of cure. But I think Lebert's statement is true, at any rate of those cases in which definite symptoms have been produced by it, viz., that it is likely to prove fatal in from six to twelve months. But it has been already stated that slight degrees of ulceration may exist without any marked symptoms at all. Speaking generally, tubercular laryngitis, when it is at all marked, materially shortens life, and greatly increases its misery. It is therefore rightly regarded as a very grave complication.

Acute miliary tuberculosis.—*Acute general miliary tuberculosis* is not a common complication of phthisis, but when it occurs it is invariably fatal. It is more often found in phthisis *post-mortem* than diagnosed during life.

In my own series of fatal cases it occurred in 2·5 per cent.

Tubercular meningitis may either develop alone, or as a part of general tuberculosis.

In my series of fatal cases it was met with alone in 1·5 per cent., and in 1 per cent. more as a part of general tuberculosis.

Acute pulmonary tuberculosis.—It is in the lung itself that acute miliary tuberculosis is most frequently found, and it is not rarely the actual cause of death. In general tuberculosis the lungs are, of course, involved, as the other organs are, and they are usually the chief seat of lesion; but they may also be the only organs in which recent tubercle occurs, and in some instances are found so stuffed with recent granulations that hardly any part remains free.

Tubercular ulceration of the bowel may exist to a high degree without symptoms, and the ordinary symptoms, viz., diarrhœa and pain, may exist without ulceration, so that the diagnosis is not easy. It may lead to other complications, *e.g.*, matting together of the intestines and its results, acute or chronic tubercular peritonitis, or to perforation with diffuse or localised suppuration.

Acute tubercular peritonitis is extremely rare in the course of phthisis, unless it occur as a part of general tuberculosis. *Chronic tubercular peritonitis* also is far from common as the result of phthisis. In many cases it is antecedent to the phthisis and the probable cause of it.

Acute, simple or *suppurative peritonitis* is almost invariably secondary to lesions within the abdomen, *e.g.*, intestinal ulceration or perforation.

Perforation of the intestines is rare. Its frequency is probably less than 1 per cent. The usual seat of perforation is the small intestine, but it may be found in the cæcum, rectum, colon, or vermiform appendix.

[1] Barthez and Rilliet, *Mal. d. enfants*, vol. iii. Flint, *Phthisis*, p. 128. *Cf.* Wilson Fox p. 817.

Fistula.—Phthisical persons are liable to fistula equally with those who are not phthisical, and therefore we should expect to find the two affections accidentally associated together in a certain number of cases; but as tubercular disease of the rectum may now and then end in fistula, the percentage liability to fistula would be somewhat increased. How great this increase is it is difficult to determine, but it cannot be much; for though Allingham's figures show the presence of phthisis in 13 out of every 100 of his cases of fistula, the statistics of phthisis yield a very much smaller percentage, and this percentage, it must be remembered, includes fistula of simple, as well as tubercular, origin.

Thus Spillman places the percentage as low as 2. Sir Douglas Powell places its frequency at something under 5 per cent., and the combined figures given by Wilson Fox yield a percentage considerably under 1. In my own 326 *post-mortems* of phthisis there was not a single instance of fistula, and I think the experience of the bedside would hardly place the frequency of fistula in phthisis appreciably above that of fistula in other sick persons.

The practical conclusion to be drawn, then, is this, that even in phthisis fistula is most likely to be of simple origin, and inasmuch as the tubercular cases are associated with tubercular ulceration of the rectum—and this can generally be detected by digital examination—the cases unsuitable for operation can usually be eliminated. This being done, the ordinary operative measures may be undertaken with almost the ordinary prospects of success unless the general condition of the patient gives some contra-indication. These *a priori* considerations are borne out by practical experience, and Allingham[1] states that he does not consider phthisis, as such, incompatible with successful operation, if the general health and other conditions of the patient are not unfavourable. To this it may be added that there is always a risk in a phthisical patient of a simple fistula becoming tubercular by secondary infection, and this would be another argument in favour of early operation.

Amyloid disease, which is rapidly becoming a rare disease in general, is now most commonly seen as the result of phthisis, but its percentage in phthisis is not high, not being above 6 per cent. of fatal cases. In most of these it is discovered only on *post-mortem* examination, having given no signs of its presence during life. The diagnosis of amyloid disease depends (1) upon the history or signs of some disease capable of exciting it, *e.g.*, prolonged suppuration, phthisis and syphilis; of these phthisis is the most frequent cause: (2) upon the evidence of the affection of other organs, *e.g.*, enlargement of the liver and spleen, albuminuria or dropsy, and chronic diarrhœa. The diagnosis during life is far from easy in any case, and is often impossible.

With the removal of the exciting cause, it is possible that the amyloid change may disappear; so it is conceivable that amyloid disease may resolve in cases of phthisis which have become chronic or stationary, but I do not know of any conclusive instance of such recovery being recorded.

When the symptoms seem sufficient to justify the diagnosis of amyloid disease, the duration of life is likely to be short.

The last group of complications consists of affections which are not peculiar to phthisis, but occur in the later stages of many other exhausting and chronic diseases, viz., profound asthenia, dropsy, thrombosis, etc.

Dropsy, if not consequent on amyloid disease, is in most cases due to cardiac failure. It rarely passes beyond the legs, or reaches that extreme degree so frequent in morbus cordis. It is a bad sign when associated with an advanced case of phthisis, and foreshadows an early fatal termination. In slighter cases it may be a transient symptom only.

[1] *Dis. of Rectum.* *Cf.* Herbert Allingham, *Brit. Med. Jour.*, 1890, April 13.

Thrombosis develops, with but few exceptions, in the large veins of the leg, and, as a rule, in patients who are confined to bed. It is not altogether uncommon, but, though a late symptom, does not necessarily shorten life.

Bed-sores form only late in the disease and in bed-ridden patients. They are, for the most part, avoidable complications, and result from want of proper care and attention; if neglected, they may be the cause of death.

Miscellaneous Complications.—I have seen a patient with loss of power in his left arm, which was obviously a *pressure palsy*, the result of his constantly lying on that side; and, of course, *œdema from pressure* on the veins is far from uncommon.

Another complication may be mentioned here, uncommon but not altogether rare, which often causes great difficulty in diagnosis. I refer to *superficial abscesses* in the chest-walls. I have seen several instances of this, and in most the diagnosis from pointing empyema was not easy. They come without obvious cause, and, as a rule, without pain, and are often discovered by accident as it were, that is to say, only after they have reached a certain size. For the most part they are small; the largest I have seen was not bigger than a hen's egg. They are soft and fluctuating almost from the first, without heat, and at first without superficial redness of the skin. Their usual seat is in the lower axillary region, but they may occur in front or at the back, but always, I think, in the lower part of the chest. When incised, they yield a small amount of curdy pus, and though they rapidly contract, leave a troublesome fistula, which runs the ordinary course of a tubercular fistula. The abscesses are no doubt tubercular. When the skin becomes red and the spot tender, especially if there be physical signs in the chest near, the diagnosis is obviously difficult; but in most cases the course proves the abscess to be unconnected with the chest. There may be more than one. A man of 25, with phthisis of both upper lobes, had two in unusual places, one at the lower end of the manubrium sterni, and another under the nipple, both on the right side.

THE RELATION OF PHTHISIS TO OTHER DISEASES.

Phthisis being so common a disease, will, of course, be sometimes associated incidentally with other affections. Whether, as regards any given disease, the frequency of association exceeds that due to accidental coincidence cannot, as yet, be determined by figures, and remains a matter of speculation.

Much of the interest in this question rested, in times past, upon the belief that phthisis depended upon a peculiar modification of inflammation or upon vicious nutrition. Now that phthisis is known to be a germ disease, the influence of any given disease associated with it will depend upon the extent by which it may diminish the constitutional resistance offered by the patient to the attack of the bacillus. Indirectly any illness will also increase the actual risk of infection, the invalids being confined within doors or in hospital wards, and fed largely upon milk, possibly from contaminated sources.

The relation of phthisis to tubercular disease elsewhere has been already referred to, and it has been seen that Louis' law holds good, at any rate for the adult, viz., that with tubercle elsewhere, if any other part of the body become affected, the lungs hardly ever escape. Thus the relation of phthisis to tubercular lymphatic glands, caries of the bones and joints, etc., need not be further considered, except to state that these affections show the tendency which tubercle has to remain a local disease for a long time, if not for life. But they also show that tubercle anywhere is a constant source of danger, and that very trifling causes may excite either a fresh outbreak of the disease locally or the infection of other parts of the body.

Tuberculosis following injury.—In this respect it is interesting to bear in mind the numerous instances recorded in which a slight injury or operation has been followed by tuberculosis.

Many cases have been recorded in connection with the testicle, spine, and joints in which an injury has apparently been the starting point, and even tubercular meningitis has sometimes appeared to follow a blow or fall on the head. In the case of the lung, so many instances

are now on record that the sequence of events cannot be denied. The cases fall into two groups—(i) that in which the patient was already phthisical before the injury, and (ii) that in which he was apparently healthy. In the former group all that the injury did was to start the disease into fresh activity, and it is to this group that the majority of recorded cases belong. Even in the latter group, in some of the cases *post-mortem* examination proved the existence of lesions antecedent to the injury, and in them the injury had caused an unusual amount of hæmoptysis, which of itself rendered the existence of some old lesion probable. In a case quoted by Fagge (p. 993), although phthisis appeared to follow the injury, and the lesion produced by it was tubercular, still the patient had chronic phthisical lesions in both apices.

In the residue of cases in which, as far as could be judged, the patient was healthy till the injury, and became phthisical after, it is clear that the lesion produced by the injury on the lung must have become infected with tubercle bacilli, and they must have been either introduced from without by the air inspired, or have been present in the body, perhaps in the blood, the lesion determining, as in other cases, the seat of development. The two most important contributions on this subject are Lebert's article and a paper by Mendelssohn, partly bibliographic and partly clinical, in the *Archiv fur klin. Medicin*, vol. x.

In discussing the relation between phthisis and any other disease, we may consider

1. The effect of the disease in question upon persons presumably not previously tubercular in rendering them more susceptible to phthisis. Diabetes mellitus, for instance, appears to have such an influence.
2. The effect of the disease in question upon pre-existing phthisis. Thus influenza not only predisposes to phthisis, but, when occurring in persons already phthisical, seems to rouse it into fresh activity.
3. The effect of phthisis upon the disease in question.

Theoretically any local disease of the respiratory organs might predispose to phthisis, either by virtue of the diminished tissue resistance caused by the disease, or because of the interference the disease produces with the normal respiratory functions, by which the invasion of the lung by the germs is favoured, or their expulsion rendered more difficult. In this way abrasion or ulceration of surface would favour infection, while pleurisy or any other affection of the lung, which impaired the movements of the chest, would render expulsion more difficult.

There are three affections of the respiratory organs which deserve especial consideration, viz., pneumonia, bronchitis, and pleurisy.

Croupous Pneumonia.—There is no evidence to show that croupous pneumonia renders the patient in any way more susceptible to phthisis. In 100 consecutive cases of acute pneumonia which were not fatal, none showed signs of tubercle in the lung, and, in the same way, of 100 consecutive *post-mortem* cases, there was not a single instance among them of recent tuberculosis.

Where pneumonia occurs in the course of phthisis, the danger of the attack is, of course, greater, but it frequently runs its ordinary course, and ends in recovery, while some patients even have several attacks, the phthisis meanwhile running its own course unaffected for good or ill.

The cases in which what appeared at first to be an ordinary attack of acute pneumonia, ends in phthisis, admit of two interpretations:

1. Either that the acute pneumonia ended in phthisis, *i.e.*, that it led to or caused the tuberculosis;
2. Or that the pneumonia was tubercular from the commencement.

The first explanation is very difficult of proof, and the probabilities are all strongly in favour of the second. There are certainly cases of phthisis which begin so acutely and with so much inflammatory consolidation as to resemble acute pneumonia; but as a rule they present peculiarities from the beginning,

the temperature being more oscillating and the duration of the fever longer. Microscopical examination of the tubercular lungs has shown that tubercle can excite any degree of inflammation, even to the production of a fibrinous exudation, indistinguishable from acute pneumonia.

The probabilities are therefore all in favour of the view, that in those cases, in which phthisis has apparently commenced as croupous pneumonia, the pneumonia has been tubercular from the onset.

Broncho-pneumonia.—The difficulties of determining the relation of simple broncho-pneumonia to phthisis are still greater, for it is just this form of patchy consolidation which is ordinarily produced by tubercle. In the adult broncho-pneumonia is almost invariably tubercular, but in children often not, as far as can be judged, though the difficulties of diagnosis, except from the course the case runs, may be very great.

Bronchitis.—Bronchitis and phthisis, being both of them such very common diseases, must of necessity be not infrequently associated, yet we have no evidence that bronchitis ends in phthisis as often as we might expect. Indeed, we seem almost justified in drawing the opposite conclusion, viz., that bronchitis is in some respect antagonistic to phthisis. If this be so, the explanation may be that the profuse secretion in the tubes entangles the tubercle bacilli introduced with the air, and thus prevents their gaining access to the lungs. When phthisis does follow bronchitis, it is, as a rule, only in the chronic and long-standing cases in which the bronchitis has produced secondary changes in the lung, such as bronchiectasis or fibrosis.

The relation of acute bronchitis to tuberculosis is much more difficult to settle. It is usually impossible to say, in any case in which phthisis has appeared to follow bronchitis, that the bronchitis was not itself of tubercular origin. For acute general bronchitis may be the earliest evidence of acute tuberculosis of the lung, while a localised bronchitis is almost always evidence of some local lesion, and that lesion is most likely to be tubercular, especially if it occur at the apex.

If bronchitis occur in a phthisical patient, it of course aggravates the danger, and may, in an advanced case, be the cause of death, and if it often recur, it seems to accelerate the disease.

Pleurisy.—Pleurisy is in some cases no doubt simple, *i.e.*, not tubercular, and not leading to phthisis; yet, on the other hand, many of the cases which have appeared to be simple and to have completely recovered, turn out to be tubercular, even though they do not lead to phthisis; lastly, pleurisy may be the first sign of a commencing tuberculosis of the lung. The difficulties, therefore, are chiefly those of diagnosis and are often insuperable.

So long as a dry pleurisy is limited to the axillary region, or to the base of one lung, it is probably of non-tubercular origin; if it be localised over the upper lobe, it is almost certainly tubercular; and if general, *i.e.*, involving the whole of the side, both upper and lower parts, it is in all probability tubercular. Again, if dry pleurisy occur on both sides, even if it be at the base only, it is almost certainly secondary to the same lesion affecting both sides, and that lesion will in most cases prove to be tubercular.

On the other hand, we know that phthisis is very likely to cause pleurisy; in fact it generally does, for in a case of advancing phthisis, the lymphatic tissues beneath the pleura are the favourite seats of the secondary tuberculosis, and this may excite any degree of inflammation in the pleura, exudative or not. Usually the pleurisy is non-exudative, and leads to fibroid thickening, with obliteration of the cavity. It spreads, as a rule, slowly from the neighbourhood of the phthisical lesions in the lung, and hence it is that dry pleurisy of the upper lobe is so suggestive of its nature.

If effusion occur, the fluid may be serous, sero-purulent or purulent. If, in a case not obviously phthisical, a serous effusion develop with great rapidity, and the fluid, when drawn off spontaneously, coagulate or be blood-stained, it is very likely to prove to be due to tubercle and end in phthisis; and, strange as it may seem, this probability is increased, if the effusion also disappear with unusual rapidity after tapping.

Dry pleurisy does not materially affect the prognosis of phthisis. Patients suffering from it may completely recover, and if it leads to adhesion, this is, as far as it goes, a conservative process, and diminishes the risk of pneumothorax. Serous effusion, in the same way, may completely resolve and not recur. Empyema, however, in phthisis is always a very grave complication.

The opinion that pleuritic effusion checks the progress of phthisis is, I believe, based more upon theory than observation. I have seen phthisis apparently stationary while the effusion lasted, and progress after its removal or disappearance; but, on the other hand, I have more frequently seen it continue to advance uninterruptedly, or even at an increased rate, so that it is clear no rule can be laid down. If there be any truth in the theory at all, it is but a partial truth, and if applied in practice, is more likely to do wrong than right.

Hæmoptysis (*Phthisis ab Hæmoptöe*).—A person apparently in good health may be suddenly attacked with hæmoptysis, immediately after which phthisis becomes manifest and runs its usual course. This represents a distinct clinical group of cases, which have been described by the term *phthisis ab hæmoptöe*, it being assumed that the patient became phthisical because of the hæmoptysis.

This theory rests upon three assumptions:

1. That the patient was really perfectly healthy before the hæmoptysis.
2. That, as necessarily follows, the bleeding came from a previously healthy lung.
3. That blood in the lung is of itself capable of causing phthisis. If these assumptions can be shown to be untrue, the theory fails.

The evidence that blood, as such, is incapable of producing phthisis is overwhelming. Contusions, lacerations, wounds of the lung of every kind, show that blood may be freely effused into the lung and yet be completely absorbed, and produce no lesion whatever, and if any lesion result it is some form of ordinary inflammation which runs its usual course and does not end in phthisis. Blood is, as a matter of fact, as readily absorbed from the lung as water, and is no more likely than water, if pure, to produce lesions of any kind.

Even in phthisis itself the hæmoptysis seems often to do good rather than harm; the blood is, at any rate, readily absorbed. When the hæmoptysis is profuse, the blood rarely clots in the lung, and, when it does, forms the ordinary kind of clot which is after a time expectorated, and does not, as has been asserted, caseate. There are a few instances in which a recent clot has been found to be infiltrated with tubercle bacilli, but none in which the clot has been shown to become caseous.

We cannot, therefore, avoid the conclusion that blood as such cannot produce phthisis.

Nor is the second assumption true, that free hæmoptysis can occur in a healthy person from a healthy lung, with the rare exceptions in which bronchial hæmorrhage follows violent expiratory efforts or straining. For it is proved beyond question, first that the bleeding is due to gross lesions of large vessels in a chronic cavity in the lung; secondly, that such chronic cavities may exist without any evidence of their presence, so that the person would be regarded as perfectly healthy; and lastly, that these cavities may be quite undiagnosable by physical examination.

The proof of each of these statements is given in the section on Hæmoptysis.

It follows, therefore, that we are in a position to deny every one of the assumptions upon which the theory of phthisis ab hæmoptöe rests, and the theory must therefore fall.

Even on its own merits the theory is unsatisfactory, for when the blood is effused, even if it come from a lesion in the apex, it gravitates to the base, and often yields evidence by physical signs of its presence there; yet when phthisis develops after hæmoptysis, it shows itself in its usual place, viz., at the apex and not at the base.

The facts are, therefore, in direct opposition to the theory of phthisis ab hæmoptöe. On the contrary, the hæmoptysis, so far from producing the phthisis, is itself evidence of the pre-existence of tubercular disease. That phthisis does not more frequently follow, or progress more rapidly after, hæmoptysis, is to be explained by the fact that the hæmorrhage comes from a chronic cavity, in which the tubercular process may be no longer active, and in which, sometimes, tubercle bacilli are no longer to be found. When phthisis does follow hæmoptysis, it is due not to the blood, but to the tubercle bacilli, which have been carried by the blood out of the cavity whence the bleeding came, and so disseminated throughout the lung.

Syphilis.—There is certainly no special liability to phthisis among syphilitic persons. Chronic syphilis, like chronic tuberculosis, may lead to fibroid change in the lung, and in an advanced stage there is nothing in the lesion by which its real nature can be determined. Gummata may occur in the lung, and may possibly break down, but I do not know of any conclusive case which has proved this to happen. In most cases syphilitic lesions in the lung are characteristic, and are not such as are likely to be confused with those of tubercle. It is, of course, impossible to say that there cannot be such a disease at all as syphilitic phthisis; but it is, at any rate, certain that much of what has been called by that name is nothing but ordinary tubercular phthisis occurring in a syphilitic person; and considering the frequency of the two diseases, the rare association of the two together is striking, and suggests an antagonism rather than a relationship between them.

Morbus Cordis.—The theory that caseation depended upon defective blood supply led to the belief that such diseases of the heart as were attended with pulmonary congestion would be antagonistic to phthisis. I think there can be no doubt that phthisis and morbus cordis are not as often associated as the frequency of the two diseases would lead us to expect, yet they occur together sufficiently often to show that there is no necessary antagonism between them. Speaking generally, the association is met with in about 5 per cent. of all cases of phthisis.

My own cases yield 8 instances out of 200, *i.e.*, 4 per cent.

Kidd's,[1] 27 in 500, *i.e.*, 5·4 per cent.

Osler's, 12 in 216, *i.e.*, 5·6 per cent.

Of the different forms of heart diseases, the association is rarest with mitral disease, especially mitral stenosis, and this is the more striking as mitral diseases are most frequent in early life, that is, at the period when phthisis is also most common.

Of my own 8 cases, 3 had disease of both aortic and mitral valves; 1 of the mitral only, and 2 of the aortic only. In the other 2, pericarditis was the lesion, in one recent and in the other chronic.

[1] *St. Barth. Hosp. Rep.*, xxiii. 239.

Fagge stated that mitral stenosis was an almost complete bar to phthisis—the *post-mortem* records of Guy's supplying only 4 cases in the course of thirty years. Kidd's cases give one instance only in 500 cases ; Walsham's [1] also one in 130 cases.

Tubercular endocarditis has been described, but the observation requires confirmation.

In congenital diseases, on the other hand, the association is more frequent, so that pulmonary stenosis is said by some writers actually to favour phthisis. Lebert states that 30 per cent. of the cases of pulmonary stenosis die with tuberculosis of some form, though not necessarily of the lungs only.[2]

In a similar way the pressure of an aneurysm upon the vessels at the root of the lung was supposed to predispose to phthisis. Statistics vary greatly, and the association is probably accidental, but it must be borne in mind that pressure on the vessels, whether by aneurysm or new-growth, may cause necrotic changes in the lung, which, though destructive, are not necessarily tubercular.

Cancer.—The two diseases prevail at different periods of life—phthisis early and cancer late in life. In persons who die of cancer, old tubercular lesions are not uncommon, but do not occur with greater frequency than in persons of the same age dying of other diseases (41 per cent., K. Fowler). In a few instances recent tuberculosis has been found. Lebert met with as many as 9 out of 101 cases. He also showed that out of 79 mothers who died of cancer, 37 had tubercular children, and, again, that 75 per cent. of tubercular persons had cancerous antecedents. The explanation is probably this, that the persons inherit or transmit a condition of feeble tissue resistance, so that they fall easy victims to the attack of the germs of tubercle in the one case, or of cancer in the other, or may even possibly become affected with both.

The statement, that it is commoner in cancer-cases to obtain a family history of tubercle rather than cancer, is but another way of stating the fact that tubercle is so much commoner a disease than cancer.

Vaccination.—There can be no doubt that careless vaccination may lead to the inoculation of tubercle as it may of syphilis or erysipelas. It may be also assumed that vaccination from the arm of a child already tubercular is not without risk.

Barthez and Rilliet quote figures to show that the liability to tubercular affections is greater among vaccinated than among unvaccinated children, but they do not attribute it directly to vaccination.

The probabilities are that all risk can be avoided by proper aseptic precautions, and that cases of infection with tubercle, as with other diseases, are due to the neglect of such precautions, so that the occurrence of tubercle after vaccination is condemnation, not of vaccination, but of the vaccinator.

Feeble Health.—There can be no doubt that feeble health predisposes to infection of all kinds by reducing the resistance of the body. In this way anæmia, and all affections which lead to it, such as chlorosis, chronic dyspepsia, exophthalmic goitre, rickets, etc., may lead to tubercular disease, but in many cases it may well be that the anæmia has been not pre-tubercular, but the early signs of an already existing tuberculosis.

So, again, where the health has been impaired, as during convalescence from some acute illness, *e g.*, a fever, or by pregnancy and lactation, phthisis may develop.

Specific Fevers.—After most of the specific fevers phthisis is rare, but there are three which stand in a closer relation to phthisis than the rest, viz., measles, typhoid fever and influenza.

[1] *Brit. Med. Journ.*, Oct. 28, 1899.

[2] Wilson Fox, p. 922, gives a review of the literature of the subject.

Measles.—The bronchitis and broncho-pneumonia which are such common complications end occasionally in phthisis.

This occurs, according to Wilson Fox, in 4·4 per cent. (14 times in 314 cases). Barthez and Rilliet [1] give a percentage of 9, and others a still higher percentage.

Typhoid Fever.—Typhoid fever may rouse up into fresh activity a pre-existing phthisis, and thus seem to cause it. This is, I believe, the more usual occurrence. The percentage of cases in which phthisis follows typhoid is distinctly small, and phthisical patients seem to be, as Rokitansky stated, singularly little liable to typhoid.

The difficulties of diagnosis, however, as between typhoid fever and phthisis are sometimes very great, and may be insuperable except by the course the case runs.

For, on the one hand, some cases of typhoid are associated with so much bronchitis, and even purulent bronchorrhœa, that the real nature of the case is masked by the predominance of the chest symptoms.

In a case under my care, acute bronchitis with a rise of temperature set in during the second week of convalescence from typhoid, and the question of acute phthisis was raised; but the attack proved to be only a relapse of typhoid.

On the other hand, acute phthisis may set in with abdominal symptoms and so much fever, that the diagnosis of typhoid may be made.

In a case of this kind the diagnosis had been made first of pneumonia, then, on account of the duration of the fever, it was thought to be typhoid; it ultimately proved to be phthisis, of which the patient shortly after died.

Again, in those cases of protracted typhoid, which are usually explained as due to delayed cicatrization of the ulcers, the continued hectic state may lead to the diagnosis of tuberculosis.

In a young man, for more than three months after the disappearance of all signs of typhoid, the temperature continued raised and of the hectic type, so that the diagnosis of tuberculosis seemed probable; yet in the end recovery was complete, and no signs of tubercle ever developed.

When the two diseases are associated together, they do not appear to have any special effect, either on the other.

Influenza.—There seems to be no disease of this class which has so pronounced an effect upon existing phthisis as influenza. There is no evidence that it takes any part in producing phthisis, except in a general way as the result of the ill-health it leaves behind, but there can be no doubt that it frequently rouses into fresh activity phthisis which has become quiescent. Whether the patient has been previously tubercular or not, instances have been not uncommon during the recent epidemics in which the patient was either in good or fair health until the attack of influenza, but never well after it, and died before long with phthisis. Although some of these cases, after beginning acutely, become chronic, as a rule they run a rapid course.

A patient died of acute phthisis within three months from being in active work, and in perfect health, as was believed; his illness commencing with a not very severe attack of influenza.

The diagnosis is in these cases sometimes very difficult, for influenza stands in close relation with pneumonia, and in some of these cases the pneumonia is rather of the broncho-pneumonic type, and attended with occasional hæmoptysis. The presumption may appear in such cases to be strongly in favour of phthisis, and yet with rest and care recovery is complete.

[1] *Cf.* Wilson Fox, p. 542.

I met with an instance of this in the first year of the epidemic, in a young man who, after a moderate attack of influenza, got well enough to go about his work, but his temperature remained high, rising to 102 or 103 in the evening. He lost flesh and strength, sweated at night, and spat a little blood two or three times in the course of a month. No bacilli were found, and the case was thought to be an irregular influenza-pneumonia. A couple of months' rest and change restored him to perfect health, and he has remained well ever since.

Pregnancy.—Phthisis, unless advanced, has little effect on conception or on pregnancy. Procreative power is said to be somewhat above the average in phthisical women, and below the average in man, but sexual appetite is not much impaired, except in the last stages of the disease. When pregnancy occurs, the child is usually carried to the full time and born in the natural way. Abortion rarely occurs unless the phthisis be advanced, or associated with very high temperature and violent coughing. When artificially induced it often produces more harm than if the pregnancy had been permitted to run its own course.

The child is seldom, if ever, born tubercular, though it is very likely to become so later, especially if nursed by its mother. Phthisis is one of the evils to which rapid child-bearing is supposed to lead, and it may be so, but only as the result of the exhaustion it causes.

That child-bearing has some influence upon phthisis is probable from the sudden increase in the female mortality as compared with the male between the ages of 20 and 35. It has been also shown that phthisis is more prevalent among married women than among unmarried, in the proportion of nearly 3 to 2.[1]

Pregnancy has generally a disastrous effect upon phthisis, especially if it be in active progress at the time. Though it occasionally happens that a woman may, during the early months of pregnancy, seem to be in better health than usual, still, as a rule, in the late months, and especially after parturition, the disease makes active progress. When a phthisical woman marries, she may bear the first child, and even suckle it for a time, without suffering seriously, but with each succeeding child her health becomes worse and worse, and probably after the second or third child she dies. Still she remains fecund almost to the last, and even if pregnancy occur during the last stages, she will probably live to give birth to the child.

Parturition is, as a rule, uneventful, and though it might be thought that the violent straining might cause rupture of the lung, hæmoptysis, or pneumothorax, such events are extremely rare.

Lactation.—It is after parturition, and especially during suckling, that the phthisis becomes aggravated. The risk to the child of being suckled by a tubercular mother has been already referred to, and it is best both for mother and child that suckling should be prohibited.

Hyperlactation introduces a difficulty sometimes in diagnosis, for a perfectly healthy woman, exhausted by prolonged lactation, may develop loss of flesh and strength and night sweats, and if, as is likely enough, she happen to have also a little cough, the diagnosis from phthisis may be by no means simple. The course of the case, however, clears up all doubt, for rest, feeding up, and the stopping of nursing soon suffice to restore her to health. I have seen similar symptoms, *i.e.*, loss of flesh and strength and night sweats, produced in young and healthy men by overwork and want of air and exercise, but in the same way the suspicion of phthisis was entirely dispelled by rest and change.

Menstruation.—One of the earliest effects of commencing phthisis is irregularity, scantiness, or suppression of the catamenia; or, if phthisis develop

[1] *Cf.* first Brompton Report, Reginald Thompson, "Family Phthisis."

before puberty, the catamenia may not appear at all. Women usually attribute all subsequent ailments to catamenial disturbance, and it is but natural to expect that phthisis should have been often referred to this cause. Phthisis, however, produces no more catamenial irregularity than do other diseases which lead to similar cachexia. In this relation the question of *vicarious menstruation* becomes of interest, but there are few cases of so-called vicarious menstruation which stand close investigation, and, in the absence of any conclusive evidence, I greatly doubt its real existence. If periodical hæmoptysis occur coincident with the catamenia, it should be referred to tubercular disease of the lung, as are other cases of hæmoptysis. All that has been said about phthisis ab hæmoptöe applies with equal force to vicarious menstruation, and even if vicarious menstruation were established as a fact, which I do not think it ever will be, it still could play no part in the causation of phthisis.

Rheumatic Fever.—Though rheumatic fever and phthisis are most prevalent at the same period of life, they are but rarely associated. Statistics vary a good deal, but in most the distinction is not clearly drawn between acute rheumatism, *i.e.*, rheumatic fever and the various chronic affections which are called by the general term chronic rheumatism. Pollock's figures yield a percentage of 1·5. The association of phthisis with chronic rheumatism is obviously accidental. With rheumatic fever the association is so rare that it is difficult to avoid the conclusion that a real antagonism exists between the two diseases, and this is confirmed by the comparatively rare association of phthisis with morbus cordis, and especially with that form which is the most frequent result of rheumatic fever, viz., mitral disease. Even where transient joint swellings do occur in the course of acute phthisis, it does not follow that they are due to the rheumatic fever poison; for joint swellings are not uncommon after the injection of tuberculin, and though at first thought to indicate some latent tubercular disease, they are now more correctly regarded as being of the same nature as those met with in other septic conditions; still, so far as I know, it has never been suggested that they were rheumatic.

Gout.—Phthisical patients are hardly ever gouty, and this may in great part be explained by the fact that the two affections prevail at different periods of life.

Plumbism, though once thought to be antagonistic to phthisis, is without influence; phthisical persons are affected by lead, like other persons, and those who suffer from chronic lead poisoning may become phthisical.

Renal Disease.—There is only one form of renal disease which stands in any close relation with phthisis, and that is *amyloid disease*. My own statistics show that amyloid disease occurred in 6 per cent. of deaths from phthisis, and in the great majority of them the kidney was involved.

Granular kidney occurred in 1·5 per cent., which is probably below the average required for accidental association. The same result was arrived at by Dickinson from the other side, for he found the percentage of tubercular lesions in granular kidney was below the general average. When renal disease is associated with phthisis, it appears to have no special effect upon the course of the disease, unless it be to keep the general level of the temperature lower than it would otherwise be.

Lupus.—The relation of lupus to phthisis has become of great interest since the discovery in lupus of a bacillus which is undistinguishable from that of tubercle, so that lupus is often regarded as tuberculosis of the skin. If this be true, it is strange that lupus is so rarely followed by tuberculosis of the lymphatic glands, by phthisis, or general tuberculosis. It is true that now and then patients are found who have both lupus and phthisis, but not more

frequently than we should expect as the result of accidental association, considering that both diseases are most frequent about the same period of life. On the other hand, lupus is certainly a very rare complication of phthisis, *i.e.*, phthisical patients rarely develop lupus. When, however, the skin is secondarily infected in the course of phthisis, or primarily by inoculation, it is by no means so uncommon to find the lymphatic glands and the body become infected, and the patients die with tuberculosis elsewhere. Clinically we can hardly avoid the conclusion that, in spite of the resemblance between the bacilli, the two diseases are not identical.

If they are the same, the differences must be due to local conditions, which check the growth and development of the bacilli. Perhaps, as Payne suggests, the temperature is the chief factor in the case. The tubercle bacilli grow best at about 100°, but the skin temperature is usually much below this, by 10 or even perhaps 20 degrees. Although inoculation-tuberculosis differs in many respects from lupus, still it resembles it in the slowness with which the disease spreads locally.

The same peculiarity is also observed in some new-growths of the skin, for example, cancer, and especially sarcoma. Mr. Hutchinson recently exhibited a case of sarcoma of the skin of the abdomen of twenty years' duration.

Alcoholism.—Alcoholism and phthisis are both such common affections that they must of necessity be not infrequently associated by way of accident; but any closer relation between them it is difficult to prove, and statistics vary greatly.

Cirrhosis of the liver is not common in any series of cases of phthisis.

My own *post-mortem* figures yield a percentage of 1·5. Leudet's percentage is somewhat higher, viz. 3. From these must be deducted an indefinite number in which the patients become alcoholic after they are phthisical. For it is not uncommon to find phthisical patients develop habits of drinking, either because of a belief that stimulants are necessary in order to keep up their strength, or because advised by their doctor to take them.

If, on the other hand, a series of alcoholic cases be taken, it is true that a good number of them die of phthisis.

Thus Leudet found that on the average the percentage was 12·5, and in extreme cases as high as 17. Still, this is not much above the general death-rate from phthisis.

Whether phthisis be, or be not, actually more prevalent among alcoholics than among temperate persons, there can be no doubt that when phthisis attacks an alcoholic person it is likely to run an acute course. Thus Hector Mackenzie records 75 fatal cases of phthisis in drinkers, and in 46 of them a cirrhotic liver was found. In 82 per cent. of these the lungs presented the lesions of acute phthisis. It has been held that alcohol produces cirrhotic changes in the lungs like those met with in the liver, but this has been shown to be an error, and the fibrous changes, when they occur, are of tubercular origin. On the other hand, the experience of alcoholic peripheral neuritis renders it not unlikely that the liability to phthisis is actually increased in alcoholism; for no small number of these cases die of phthisis, and in them also the phthisis tends to be of an acute form, and to run a rapid course.

Nervous Diseases.—The first of these is *insanity*, especially in its chronic forms, and in those attended with depression (*phthisis a melancholia*). There can hardly be any connection between mental disease and phthisis other than this, that the general health is greatly impaired, so that little resistance is offered to infection. Insane persons certainly do die frequently of phthisis, and in excessive proportion, but the statistics of different authorities differ widely, and it is impossible to avoid the conclusion that the mortality is largely affected by the sanitary conditions and modes of life in the different institutions.

Phthisical persons may become insane, and probably for the same reason that patients do after other acute or long-continued illnesses. It is a rare complication, but the prognosis is very bad in respect of the mental condition, and also, though in less degree, in respect of the duration of life.

I have met with an instance of this in a man of 51, who had subacute phthisis of a few months' duration. There was no family history of insanity in his family, but he had had syphilis thirteen years previously. He went quite mad all at once, and had to be sent to an asylum, where he soon died.

Peripheral Neuritis.—It has been mentioned already that a good many cases of peripheral neuritis become phthisical and die of that disease. It is also true that peripheral neuritis develops in a certain number of cases of phthisis, though in such a way as to be frequently overlooked, the loss of power being referred to general causes.

The most complete article on the subject is one written by Pitres and Vaillard,[1] in which the literature of the subject is given, as well as records of cases of their own.

Four cases are described in which the symptoms were pains of varying intensity and loss of power in the lower extremities, and on examination the whole nervous system proved to be healthy except the peripheral nerves, which showed the lesions of neuritis.

Peripheral neuritis in phthisis occurs in three forms:

1. *Latent*, where the pains are slight or indefinite and their nature often overlooked.
2. *The amyotrophic form*, in which the muscles of all the extremities, the back, neck, abdomen, and diaphragm, may be affected, not all at once but by gradual progression. The paralysis and wasting may be well marked and sometimes very rapid, the electrical contractility greatly impaired or lost, and sensation also affected.

Attention was first drawn to this form by Eisenlohr.[2]

It is probably to peripheral neuritis that those instances of limited paralysis occasionally met with in the course of phthisis are to be referred, *e.g.*, paralysis of the flexors of the fingers (Leudet), of one arm (Perroud),[3] etc.

3. *The form in which sensory disturbances predominate*, hyperæsthesia, anæsthesia, or neuralgia.

Thus there may be exquisite pain in the joints both on touch and movement; muscular hyperæsthesia in the thighs and calves; cutaneous hyperæsthesia over the whole body, one limb, or in patches; and, lastly, neuralgias of fixed seat, especially in the sciatic, but occasionally elsewhere, *e.g.*, in the radial, cubital, crural, cervico-brachial or other nerves. Such neuralgias are very rebellious, *i.e.*, of long duration and refractory to treatment. They may be accompanied with herpes zoster. They sometimes improve spontaneously for a time, or disappear in one part to develop in another.

There is good reason to believe that peripheral neuritis will explain most of the irregular polymorphic nerve troubles which occur in the course of phthisis.

The occurrence of peripheral neuritis in phthisis tends to bring phthisis into relation with other germ or specific fevers, such as diphtheria, typhoid, small-pox, or typhus, with all of which peripheral neuritis is occasionally found associated.

Addison's Disease is now generally regarded as of tubercular nature, though it is true that patients suffering from Addison's disease rarely present signs of acute phthisis, and probably never die of it. It is this, I suppose, which

[1] *Rev. de Med.*, 1883, 193.
[2] *Centralbl. f. Nervenheilk.*, 1879, No. 5, p. 100.
[3] *Gaz. hebdom.*, 1878, p. 617.

Wilson Fox refers to when he says that the association of Addison's disease with phthisis is unknown; for Greenhow showed that if Addison's disease were associated with any other lesion in the body, it was with tubercle, and in nearly half the cases (31 out of 64) with tubercle in the lungs.

Of 7 cases examined *post-mortem* in St. Bartholomew's Hospital, the lungs were healthy in 4, in 2 there was chronic tubercular mischief at both apices, and in another general obliteration of the left pleural cavity, though not obviously of tubercular nature.

In some cases of phthisis the skin becomes so darkly pigmented as to suggest Addison's disease, without any affection of the supra-renal capsules being found after death.

Diabetes Mellitus.—It is very rare to meet with glycosuria in phthisis, or, more correctly, for a patient with phthisis to develop diabetes mellitus.

Richardson's statistics state the frequency of its occurrence as not more than 1 in 1500.

On the other hand, phthisis is a very common mode of death in diabetes, and that at any age.

Finlay and Coupland give its frequency as 11 in 20; Stephen Mackenzie 20 in 37; Frerichs 87 in 250—yielding a combined percentage of 40.

It develops, as a rule, not in the acute cases, and is rare in those which are of less than two years' standing. It usually runs an acute course, and used to be described as caseating pneumonia. Diabetic phthisis is not a special form, but is in all cases tubercular.

In 50 consecutive cases of diabetes taken from the *post-mortem* records of St. Bartholomew's Hospital, the lungs were healthy in 23 and tubercular in 21. Of these 4 presented the lesions of chronic tuberculosis, but in the rest the phthisis was active and the cause of death. One of these cases was complicated with pneumothorax, and another with empyema, and 3 others with acute pneumonia. The lung was also gangrenous in one of the pneumonia cases, and in two of the cases of acute phthisis.

Of the non-tubercular affections pneumonia occurred once; abscess, probably pneumonic in origin, also once; and empyema twice. One other case died with general pyæmia.

THE COURSE, DURATION, AND TERMINATION OF PHTHISIS.

Course.—The course of phthisis is in most cases continuous, though not uniform, *i.e.*, it advances, *saltatim*, by a series of exacerbations separated by periods of more or less complete remission. Even in acute cases these remissions are seen, and at times are so marked as to hold out hopes that recovery may occur. In the chronic cases the remissions are well marked, and may continue for some time, even months. In others, into which category most of the chronic cases come, complete intermissions may occur in which all symptoms disappear, and the disease seems to become stationary. If it remain stationary, the case is often described as cured, or better healed, phthisis, for the cure is, as in other cases of destructive lesions, by cicatrization.

The difficulty in forecasting the course of phthisis lies in the fact that the most acute case may become quiescent, and the most chronic may recrudesce.

Duration.—The duration depends upon the course the case runs. Thus we know that if the case continue acute its duration will be short; if it be subacute it may last two or three years; and if chronic many years. Thus it has been stated that phthisis may last from a few days to forty years. Such general statistical statements are of little use in practice, except as furnishing general rules, which may be some guide in estimating the probable duration of any given case.

Statistics, I think, show that the average duration of life in phthisis, speaking generally, is longer than was once supposed.

Pollock's figures yield an average of four years, while the early writers of the century put it at not more than two years (Laennec, Andral).

This is but another way of expressing the pathological fact, that tubercular lesions have a greater tendency to become arrested than was once thought, and that when arrested they are compatible with a fairly long life.

Many of the statistics published are taken from out-patient hospital practice, or, if from private sources, from what may be called current practice, *i.e.*, the duration of the disease in fatal cases is reckoned in with that of those still living. This must necessarily include an undue proportion of chronic cases, and will therefore raise considerably the general average. For this reason the average is best determined from the fatal cases, for they give, at any rate, a fixed terminus ad quem, the terminus a quo, *i.e.*, the date of commencement of the disease, being in hospital cases, at any rate, a matter of history and very difficult to determine accurately, for so many patients do not come under observation until their symptoms are pronounced and the disease some way advanced.

The following figures show the average duration of fatal cases :—

Duration.	Louis, 307 cases.	Lebert, 239 cases.	First Brompton Report.
Under 1 month,	1·3	2·3	40·8
,, 2 months,	5		
,, 3 ,,	9		
,, 6 ,,	33	18	
,, 9 ,,	53		
,, 1 year,		48	
,, 1½ ,,	86	61	
,, 2 years,		70	85·5
,, 3 ,,		80	
,, 4 ,,	14	86	
Above 4 ,,		14 ?	6·5 ?

According to these figures, out of every 100 cases 40 die within the first year, and only about 14 live more than four years.

Acute cases.—The average duration seems to be between three and six months, and to all these the name Galloping Phthisis is given, but instances of both shorter and longer duration than this are not uncommon. Fagge gives 8 cases which ended in from five to eight weeks, and Wilson Fox has seen them end in from six to nine weeks; his average for 41 cases was $5\frac{3}{4}$ months. Cases are quoted of even much shorter duration than this, *e.g.*, eleven days (Andral), thirteen days (Traube), twenty-one and twenty-four days (Chouppe). Many of these cases, I cannot help thinking, were cases of acute pneumonia or acute tuberculosis in patients previously phthisical, for even acute miliary tuberculosis of the lung rarely lasts less than four or five weeks, and in many instances even longer.

On the other hand, many factors help to prolong life, notably the condition of health at the time of onset of the disease.

For example, a lad of 17, at school, and in perfect health, attacked with phthisis in its most acute form, fought with his disease for nearly twelve months before he succumbed, although the temperature hardly ever fell below 103°, and was often 104° or 105°.

Subacute cases.—Of the subacute or more remittent forms of phthisis the duration is much longer, being on the average two to three years. It is to this group that the great majority of cases belong.

Chronic cases.—When once the disease has become arrested the duration may be very much longer. In many of the chronic cases the disease is quite stationary, and with such stationary or "cured" lesions the patient may live to the natural term of life; yet it is hardly fair to include these cases among those of the active group, and to embrace them in our estimate of the duration of phthisis, any more than we should attempt to determine the duration of syphilis by the time a patient lived with syphilitic scars. The reason why such cases are often included in statistics is, that many of these quiescent cases do ultimately die of phthisis because the disease recrudesces, or they die of some affection like hæmoptysis to which the phthisis has led. Of 1000 chronic cases (Williams), the average duration was between seven and eight years, and 8 per cent. of them lived more than twenty years.

When phthisis has once passed into the chronic or quiescent stage, the duration of life may be considerable, and the longer the phthisis has lasted the less it seems to affect the duration of life, *i.e.*, the less the expectation of life differs from that of healthy persons. The frequency with which persons who have died of other causes at the natural term, and have been found to have had healed tubercular lesions, has been often referred to, and, on the other hand, persons known to be phthisical have lived to a good old age; thus Ruhle described the case of a woman of 90 with a large chronic cavity in one lung. Still such chronic cases have risks of their own; thus Coupland records a case which died of hæmoptysis twenty-four years after all signs of active phthisis had disappeared; again, the disease may recrudesce and the patient die, years after the first onset, with the symptoms of acute phthisis; lastly, of course, the existence of damaged lungs aggravates the prognosis of any intercurrent disease.

Statistics seem to justify the following general conclusions:—

1. That a very small percentage of cases, not more than 3 or 4, terminate in less than three months.
2. That in about 40 per cent. the duration is not more than a year; that nearly one half of the cases will be dead in twelve months, and nearly three-quarters by the end of two years.
3. That of the remainder, only about 12 per cent. live beyond the end of the fourth year.
4. That only about 1 patient out of every 10 lives more than four years; though if a patient survive the fourth year, life may be considerably prolonged.

Although these general statements are true, the difficulty lies in applying them to individual cases, and it is with this problem that prognosis is concerned.

But even these general conclusions must be accepted with considerable reservation, for the mortality tables already given show not only that there has been a great reduction in the general mortality from phthisis, but that the period of greatest incidence is, roughly speaking, ten years later than it used to be fifty years ago.

Modes of Death.—Phthisical patients may die—

1. Of the disease itself.
2. Of some complication directly caused by the disease.
3. Of some intercurrent malady which would not have proved fatal if they had not been phthisical.

1. *Death due to the disease itself.*—When patients die of the disease itself, the causes of death fall, like the symptoms, into two groups—constitutional and local,

and the patients die either of *asthenia* or of *dyspnœa*. In the latter case the shortness of breath and cyanosis may be the most striking features throughout, and out of proportion to the amount of mischief in the lungs as indicated by the physical signs.

Where the constitutional symptoms are marked and the fever high, the patients may pass into what is practically a septic condition, and die in a condition resembling the typhoid state, with extreme prostration, low muttering delirium, and subsultus. Even when the fever is low, the most profound cachexia may develop, and the patients die, as those with cancer do, apparently of sheer exhaustion. Both of these conditions may be regarded as toxic states, and as the acute and chronic stages respectively of tubercle-poisoning. In all cases where the asthenia is profound, it may be greatly aggravated and the end hastened by the supervention of any complication, such as diarrhœa, pain, severe cough, and want of sleep. The actual cause of death is, no doubt, in many cases cardiac failure, for the feebleness of the circulation is often shown by the occurrence of œdema in the feet and legs, and by dilatation of the heart. Death may sometimes be sudden and unexpected, by syncope; but except in the last stages, or as the result of some sudden complication, sudden death in phthisis is very rare.

2. *Death due to some complication directly caused by the phthisis.*—Chief among these are hæmoptysis or pneumothorax.

Hæmoptysis, as already stated, is not fatal in more than 2 per cent. of all the fatal cases of phthisis; the cause of death being either suffocation, where the amount of hæmorrhage is very large, or exhaustion, where the hæmorrhage is not so large but frequently repeated.

Pneumothorax is the cause of death in about 1 per cent. of the fatal cases of phthisis. It may be immediately fatal from shock; or from sudden suffocation, owing either to the collapse of the lung or to the emptying of the cavities of the collapsed lung into the air-tubes of the opposite side. Usually death takes place more gradually after some hours or days, either from increasing suffocation or from exhaustion, which are also the usual modes of death when purulent effusion takes place.

Tubercular affections of the larynx are always grave; for they add greatly to the exhaustion by the pain, dyspnœa, and cough they occasion. They may also lead to œdema or sudden obstruction of the larynx, and prove rapidly fatal, if the symptoms be not immediately relieved by tracheotomy.

Peritonitis is a rare cause of death. It may be acute and suppurative as the consequence of the perforation of a tubercular ulcer of the bowel. When chronic, the adhesions formed may lead to intestinal obstruction.

Acute tuberculosis, when confined to the lung, causes great aggravation of dyspnœa; when general, the constitutional symptoms are severe, and the patients quickly die of asthenia, but it often happens that the prominent symptoms are meningeal.

Tubercular meningitis appears to be the cause of death in about 8 per cent. of fatal cases.

Empyema is rarely a direct cause of death, but it makes the prognosis worse, and shortens the duration of life by the exhaustion it causes.

3. *Death due to intercurrent disease.*—Phthisis gravely increases the dangers of any intercurrent malady, and especially those of the heart or lung. At the head of these comes croupous pneumonia. Reference has already been made to the frequency with which, in fatal cases of pneumonia, the lungs have been found to be the seat of previous disease, especially of a tubercular character. As an instance of another group may be taken influenza, an affection not only attended

with great risk at the time to a phthisical patient, but likely to lead afterwards to a recrudescence or aggravation of the disease.

The causes of death in phthisis are thus tabulated by Lebert:—

Collapse, *i.e.*, asthenia,	63 per cent.	77 per cent.
Asphyxia,	14 ,,	
Cerebral,	8 ,,	23 ,,
Sudden,	5 ,,	
Hæmoptysis,	3 ,,	
Peritonitis,	3 ,,	
Miscellaneous,	4 ,,	

Diagnosis.

The diagnosis of phthisis is in most cases easy. The cough, shortness of breath, and expectoration point to the respiratory organs as the seat of the disease; the loss of flesh and strength to some chronic cachexia; the hectic to some inflammatory disease; the physical signs, which indicate consolidation of the lungs with breaking down and contraction, commencing at the apex and gradually spreading downwards, render it highly probable that the disease is tubercular in nature, and the discovery of bacilli makes it certain.

Having diagnosed the disease to be phthisis, the next thing to be done is to determine its form, *i.e.*, whether it be acute, subacute, or chronic. This is done by the constitutional signs and the course the case runs. If there be rapid loss of flesh and strength, copious night sweats, and a hectic temperature, the phthisis is in the active or acute stage. If, on the other hand, the constitutional signs are absent, the general nutrition good, and the temperature not raised, the presumption is that the disease is not progressing actively, but that it is stationary or in the chronic stage.

Careful comparison of the physical signs from time to time will also show whether the disease is progressing or not, but the activity of the disease is most easily and accurately determined by the constitutional signs; for, with but few exceptions, they are marked when the disease is active, become less pronounced or disappear as the disease becomes quiescent or stationary, and reappear as soon as it takes on activity again. The physical signs show the mischief done, but the constitutional signs show that the mischief is still continuing.

Difficulties in diagnosis are likely to arise under two conditions—

(1) Where the physical signs are indefinite, and the constitutional signs can be accounted for in other ways; and (2) where physical signs are present, but admit of other interpretations.

1. Constitutional Signs Marked, but Local Signs Indefinite.—The constitutional signs are those of continued fever of a hectic character, and they might be produced by certain other diseases besides phthisis, *e.g.*, tubercle in some other part of the body than the lungs, suppuration somewhere, typhoid fever, and some conditions of septicæmia.

Typhoid Fever.—Many of the acute cases of phthisis are diagnosed as typhoid, and the distinction is not easy, for typhoid may present marked chest symptoms, and acute phthisis marked intestinal disturbance. It is more common for acute phthisis to be diagnosed as typhoid than the converse. In both diseases the onset is usually gradual without definite rigors, and the general conditions are those of a septic fever. Typhoid fever is often associated with much bronchitis, and acute phthisis in the early stages may present no definite local signs but those only of general bronchitis. The diagnosis in such cases is made by the character of the fever and by the course and duration of the case.

The fever is, as a rule, more continued and less remittent in typhoid, more hectic and with wider daily oscillations in acute phthisis; but in the later stage of typhoid, in the stage of what may be called delayed defervescence, the two temperature curves may be undistinguishable.

The course of the case in acute phthisis is, as a rule, more prolonged, *i.e.*, the fever lasts longer, and at the same time the condition of the patient is better than it would be in a case of typhoid fever of the same duration. Rapid loss of flesh and strength is, other things being equal, more in favour of typhoid fever than of phthisis.

Typhoid fever suspected to be phthisis because of its long duration.—A case, occurring in a man of 35, thought to be typhoid at the time, though the symptoms were never quite conclusive, ran on without remission into the fourth month, the temperature remaining continuously high and being of a hectic character; the patient had some general bronchitis, and lost flesh and strength greatly. At last the question of phthisis was raised, simply on account of the uninterrupted continuance of the fever, but towards the end of the fourth month the temperature fell in the usual way to the normal, and the patient got rapidly well, so that the diagnosis of typhoid was evidently the correct one, the long continuance of the case being due, I suppose, to progressive ulceration or delayed cicatrisation in the bowel. Prolonged typhoid like this is rare. Such cases are usually due to successive relapses, in one or other of which the characteristic spots or symptoms of typhoid are recognised.

It may happen that a relapse of typhoid may raise suspicions of acute phthisis.

In a young woman of about 18 years of age who had just had typhoid fever, the temperature, after being normal for three weeks, rose again gradually until it reached 103°. The fever was remittent in type; there was a good deal of bronchitis and cough; the patient looked very ill and sweated profusely. The suspicion of acute tuberculosis, following typhoid fever, had been raised, but the case ended just as relapses of typhoid usually do, and the patient made a good recovery.

Typhoid fever is sometimes overlooked at first because of the chest symptoms with which it is associated, and in such cases the diagnosis of acute phthisis may be made, though more frequently pneumonia is suspected.

A woman of 30, with fever and general bronchitis, had profuse purulent nummular expectoration so like that of acute phthisis that the sputum was repeatedly examined for bacilli, but none found. The case ultimately proved to be one of typhoid, and the patient made a good recovery. I have never seen a similar case of typhoid in which the sputum was so profuse. It might fairly be described as bronchorrhœa, for the amount reached half a pint in the twenty-four hours.

Suppuration.—As a rule no difficulty arises, for pain and swelling show the seat of mischief and point to the diagnosis; but if the mischief be deep-seated, the diagnosis may for a time be in doubt. The confusion is most likely to arise where the suppuration is in relation with the chest, *e.g.*, with a localised empyema, especially if basic, or with subphrenic abscess.

It sometimes happens that the physician is called in to decide whether the lungs are sound in a patient who has suppuration or tubercular disease of a surgical nature, and the decision may involve the question of operation.

Thus I saw a young woman with a tubercular knee-joint and marked hectic. Is she also phthisical? was the question asked. The diagnosis had to be made by the physical signs alone, and these were indefinite. The operation was performed and complete recovery took place.

The question may arise whether, if a patient be found to be phthisical, the existence of phthisis should contra-indicate operation. If such a patient were suffering from phthisis in an advanced form, it might be advisable not to operate unless the operation were imperative; but in other cases of less degree, operation is not only not contra-indicated, but is distinctly desirable. It is true that operations upon tubercular persons are sometimes followed by an acute outbreak of tubercle elsewhere, but it is also true that so long as tubercular foci exist in the body they are a source of risk, and may at any time, whether interfered with or left alone, lead to general infection. There is no doubt that, if the patient be not

phthisical, the removal of the tubercular disease diminishes greatly the risk of his becoming so. If the patient be already phthisical, still the operation is best performed unless his condition is such as to render the chances of recovery from the operation small. In such cases the operation is often followed by material improvement, not only in the general condition, but in the phthisis as well, so that it is a good general rule in such cases to operate, and to operate early.

General Debility.—Reference has been made elsewhere to cases of general debility which sometimes cause difficulty, especially when, besides loss of flesh and strength, there may be also night sweating. I have seen cases of this kind in men who have been simply overworked, and in women exhausted by prolonged lactation.

2. Physical Signs Present, but open to other Interpretation.—According as the constitutional symptoms are marked, slight, or absent, so it will be from acute, subacute, or chronic phthisis that the diagnosis will have to be made.

(1) *Acute phthisis* may set in with such acute symptoms that the diagnosis is made of *acute pneumonia*; and rightly, for it is a pneumonia, the signs of acute consolidation of the lung are present, and even the sputum may be rusty, but the pneumonic consolidation is caused, not by pneumonia germs, but by the tubercle bacillus. The subsequent course of the case shows that we have not the ordinary form of pneumonia to deal with. The fever is more remittent and continuous, and the consolidation proceeds to break down, so that any doubt which might have been felt at first is speedily removed. The most difficult cases are those in which the pneumonia is of the wandering or creeping kind, resolving in one part and extending in another. In these cases the consolidation is, as a rule, more massive, the physical signs more marked, and the extension more rapid than with tubercle.

The diagnosis from *broncho-pneumonia* is still more difficult, and often impossible at first; for acute phthisis is a tubercular broncho-pneumonia. Age, however, often gives the clue to the correct interpretation, for in the adult broncho-pneumonia is, almost without exception, tubercular, while in children it is often simple. If there be any sputum, the presence of the tubercle bacilli will settle the diagnosis. Unfortunately it is just in these cases that sputum is often absent, for little children rarely expectorate at all, and in adults with this form of pneumonia there is frequently none also.

I have seen a girl of 16 who had neither cough nor expectoration until the last week of life; and the same also in a man of 30.

One of the most puzzling forms of pneumonia to diagnose from phthisis is that which sometimes follows influenza. Here, in the adult, a subacute relapsing broncho-pneumonia may occur, with hectic flush, marked loss of flesh and strength, and sometimes even with slight recurrent hæmoptysis. Yet the sputum yields no bacilli, and the patients recover without any evidence of tubercle, then or subsequently. The difficulty of diagnosis is increased by the well-known fact that influenza does stand in close relation with phthisis, on the one hand increasing the susceptibility to it, and on the other exciting it into fresh activity when it has been previously quiescent.

With *acute basic phthisis* the diagnosis from acute pneumonia is for a time impossible, for the seat of the mischief makes it, of course, most likely that the pneumonia is of non-tubercular origin. The nature of the case is only proved by the course the disease runs. Such cases are rare. The following is a striking instance of it.

It occurred in a lady of 48, the mother of nine children, with a strong tubercular family history. Though not robust, she was a woman of great mental and bodily activity. Except for asthma, from which she had suffered occasionally from childhood, she was in her usual health until suddenly attacked by what appeared to be acute pneumonia of the left base. From the first the temperature was markedly remittent, of a hectic character, rising to 103° or 104° every evening; the loss of flesh was rapid, and the night sweats extreme. The physical signs were those of acute consolidation of the lower lobe. The fever continued from day to day, until it was clear that simple pneumonia would not account for her illness. The question of empyema was then raised, but no evidence in support of it was forthcoming. Slowly the signs of excavation appeared, but sputum was absent throughout, so that the crucial test by the examination of it for bacilli could not be made. After two or three months the symptoms subsided, and slowly all active signs of the disease vanished, but the patient was left with a cavity in the left base. She convalesced, and continued in fair though feeble health for eighteen months. Then a similar attack occurred in the opposite base which ran exactly the same course. For a long time even now there was no sputum, but at last some was obtained, and found on examination to be crowded with bacilli. From this second attack she again rallied, and though in very weak health, she was able to be up daily and manage her household with assistance for another year. At last, about three years from her first attack, the fever and constitutional signs returned, the night sweats became profuse, and a fairly sharp attack of hæmoptysis occurred. From this time the patient ran rapidly down hill. The disease spread slowly upwards, but for a long time the upper lobes remained free, and it was not until quite the last that physical signs showed themselves in the right apex. In the end, after several months of gradual failure, she died of exhaustion, 3½ years from the time of commencement of her illness. A similar case, in a girl of 16, came under my observation, but the diagnosis was less difficult because the onset was less acute. When she died the lungs presented the ordinary appearance of phthisis, except that the more advanced changes were at the base and a large cavity existed.

(2) With *sub-acute phthisis* the condition most likely to cause confusion is a *localised empyema*, especially when it is deep-seated, as, for instance, between the diaphragm and the base of the lung.

The physical signs are then very indefinite, while the constitutional signs are the same in either case. Even when the empyema yields physical signs, they may not be conclusive. The seat of the disease raises, however, a presumption in favour of empyema; for these localised empyemata occur almost without exception in the lower part of the chest, while basic phthisis is rare, so that the chances would all be in favour of empyema. In the early stages of either disease the constitutional symptoms may be marked and the sputum absent, but the very absence of sputum is so far in favour of empyema, a diagnosis which would be confirmed if the sputum appeared more or less suddenly and did not contain bacilli, while at the same time the constitutional symptoms became less severe or disappeared.

A lad of 18 had been in a condition of marked hectic for two months or more before I saw him. The physical signs were at the base, but of an indefinite character, and on the whole the diagnosis pointed to phthisis, of which there was a strong history in the family. The case proved to be one of local empyema, for the patient began to expectorate pus which contained no bacilli, and with the appearance of the sputum the fever fell and convalescence set in.

An exactly similar case occurred in a man of 32, who ultimately made a perfect recovery.

New-growth is sometimes associated with inflammatory changes in the lung and simulates phthisis. What occurs in these cases is that, owing to compression of the bronchi and the penning up of the secretion in the tubes, or owing perhaps to some interference with the blood vessels or lymphatics, a patch of the lung becomes inflamed or undergoes a process of necrosis. The physical signs point rather to empyema than to consolidation of the lung because of the occlusion of the tubes, so that the case is often diagnosed at first as empyema.

Softening may even take place in the affected parts, and a thick pus-like fluid be found there, which, however, is not pus but broken-down necrotic lung-tissue.

I had under my care a man of about 50, who had been rapidly losing flesh and strength, and looked as if he had phthisis, but no physical signs were present except defective entry of air into the whole right lung. The temperature was hectic, and rose every evening to about 102°. After a time a patch of dulness appeared in the right axilla low down, over which the voice- and breath-sounds were entirely absent. This was thought to be a localised empyema, and a needle introduced into it brought away a pus-like fluid. An incision was then made, a drachm or two of pus removed, and a tube inserted. The symptoms continued, but it soon became clear that the case was one of malignant disease. When the patient died, a cancerous growth was found surrounding the root of the right lung, and in the lower lobe were two masses of inflammatory consolidation, one of which had broken down and formed the cavity which had been incised.

(3) *Chronic phthisis.*—As there are few, or no, constitutional signs, the diagnosis has to be made by the physical signs alone. If these point to consolidation, the consolidation may be due not to phthisis only, but to *syphilitic affection of the lung*, *pneumo-koniosis*, *atrophic emphysema*, or *new-growth.*

If they show contraction or shrinking at the base, *chronic pleurisy* or *chronic interstitial pneumonia*, as well as phthisis, may account for it.

If they show excavation the cavities may be due to other causes, *e.g.*, *abscess* or *gangrene* after pneumonia, or a *suppurating hydatid.*

Lastly, there are cases in which base cavities have been confused with *pneumothorax.* This confusion can usually be avoided if attention be paid to the displacement of organs, the side being distended and the organs displaced towards the sound side in pneumothorax, while the side is contracted and the organs displaced towards the affected side in basic cavity.

PROGNOSIS OF PHTHISIS.

The three questions that present themselves for answer in any case are:

1. Is there any immediate danger to life?
2. Is there any chance of ultimate recovery; and how far will it be complete?
3. What will be the probable course and duration of the case?

These questions have already been considered in a general way from a statistical point of view. Prognosis consists in the application of these general rules to a particular case, and it is just in this that the difficulty lies, so that those with most experience have best learned the need of caution.

1. As regards danger to life.—If the case is to terminate fatally, the dyspnœa and increasing asthenia give warning enough that the end is near. Yet death is often expected long before it occurs, though the actual dying may, in such a case, be sudden; still, except in the last stages of the disease, or as the result of some sudden complication like hæmoptysis or pneumothorax, sudden death is very rare, and need not be considered as a practical risk at all.

2. As to the prospects of cure.—Perfect cure is impossible, and complete arrest used not to occur in more than about 1 or 2 per cent. of all cases. How far these results have been improved by recent methods of treatment there are not yet figures enough to prove, but there are, I think, sufficient to show that the progress of the disease may be greatly delayed.

3. As to the course and duration.—The duration of life is always considerably shortened by phthisis. What the actual course and duration are likely to be in any given case will depend primarily upon whether it belongs to the acute or chronic group; and if to the acute, upon the degree of acuteness or the severity of the disease. If the disease be acute, the duration will certainly

be short unless some remission occur. If it be chronic, life may be prolonged for years unless exacerbation occur. Remission in the one case and exacerbation in the other often do occur when there is least reason to anticipate them.

In a young man of 18 years of age the symptoms were so acute that I did not think the parents would reach home from Australia in time to see him alive, but he lingered on for months after they reached England, and ultimately recovered so as to return home, where he is now leading an active life up country, apparently well. On the other hand, I have seen chronic phthisis that had been quiescent for many years suddenly break out into fresh activity and carry the patient off in a few weeks.

Although prognosis is upset sometimes by unexpected contingencies, still, making allowance for these, there are certain general principles which guide in most cases to a fairly accurate prognosis. Chief among these are the constitutional signs, and in respect of prognosis these are of far greater importance than are the physical signs; for they show, if present, that the disease is active, and their amount forms a measure of that activity, and, if absent, that the disease is either stationary or progressing very slowly.

The Prognosis in Chronic Phthisis.—The dangers in chronic phthisis are not so much from the disease itself, which is in a more or less stationary condition, as from the lesions to which it has led, or from the extra gravity it gives to any intercurrent disease. Chief among the former is profuse hæmoptysis consequent on the bursting of a pulmonary vessel in the walls of a chronic cavity, and among the latter such acute affections as pneumonia, general bronchitis and influenza.

Among the chronic cases of phthisis the prognosis is best in those in which one lung only is diseased and the other is in a condition of complementary hypertrophy. In such cases we may conclude that the disease is probably stationary on the affected side, and that the opposite lung is healthy, for the hypertrophy does not occur when the other lung also is the seat of phthisis. Still, even in such cases the risk remains of hæmoptysis on the one hand and of recrudescence on the other. The fear of hæmoptysis is constantly present in such cases, and though fatal hæmoptysis may proceed from a very small cavity, still, the larger the cavity the greater the risk. Hæmoptysis is the one great and ever-present source of danger.

The danger of recrudescence is illustrated by the following case :—A man of 35 had an attack of acute phthisis at the age of 30, and was not expected to live for more than twelve months. The disease became arrested. He lost all symptoms, and returned to his business, which he carried on actively and without difficulty. When I saw him, not on account of chest symptoms but because he was troubled with dyspepsia, I found him with a large chronic cavity occupying the whole of the left upper lobe. For the next five years I saw him on and off for dyspepsia, and he remained throughout well and active. Then, without obvious cause, the signs of active mischief developed anew, the other lung became involved, and in a few weeks he died of acute phthisis. This patient had recovered from his first attack of acute phthisis, but died of a second after an interval of about ten years.

Pneumothorax is an unlikely complication in chronic phthisis, for the pleura is thickened and its cavity obliterated over, and for some distance beyond, the seat of disease.

Sometimes in chronic phthisis the discharges become fœtid. The influence of this complication upon prognosis depends upon its cause. If it be due to gangrene of the lung, severe general symptoms develop, and the patient rapidly passes into a septic state and dies.

More commonly, when fœtor develops in phthisis, it is due to fœtid or putrefactive decomposition of the contents of the cavities. The fœtor is then important

on account of its unpleasantness, but though so offensive to others, the patients themselves seem often to be but little conscious of it. It is a variable and often transient condition, though it may be recurrent. It may be of no prognostic importance, but I have seen patients pass into a septic state probably from absorption, and die suddenly and unexpectedly of asthenia.

Practically the chief risk in chronic phthisis, apart from hæmoptysis, is from recrudescence, and until that occur the patients may lead fairly active and happy lives, but if recrudescence occur, the disease, as a rule, runs a rapid course. Even in chronic phthisis the general condition of the patient is some guide in prognosis. Some patients, it is true, with chronic phthisis are fat, florid, well-nourished, and look the picture of health, but they are exceptions. Usually persons suffering with chronic phthisis are poorly nourished and unequal to much physical or mental effort. The more weakly they are, the more care they will require, and so far the prognosis will be so much the worse. Impairment of health, under any circumstances, whether previously it has been good, bad, or indifferent, is of importance as indicating great risk, if not the actual occurrence, of recrudescence.

The Prognosis in Acute Phthisis.—While in chronic phthisis the constitutional signs are absent or slight, so in acute phthisis they are well marked, and they serve as a measure of the severity and gravity of the case.

The constitutional or general signs fall into two groups—(1) Those of hectic fever, with night sweats, and (2) those of increasing cachexia, *i.e.*, anæmia, loss of flesh and strength. These two groups usually stand in definite relation to one another, but not invariably; thus it may happen that there may be marked hectic without cachexia, or marked cachexia with little or no fever.

Marked cachexia is always a bad sign in phthisis, even when it stands alone; and, *per contra*, diminution of the cachexia is a hopeful sign, even when not accompanied with other signs of improvement.

The cachexia may be profound or even fatal, and that without fever or evidence of sufficient tubercular disease in the lungs or elsewhere to explain it, and yet, on *post-mortem* examination, chronic tubercular lesions may be found widely disseminated through the lung, or even generally throughout the body. The lesions pathologically come into the category of chronic tuberculosis, but the symptoms are severe and acute. I suppose the condition may be regarded as chronic tubercular poisoning. It is more often met with in children with chronic tubercular glands than in patients with phthisis.

Profuse *night sweats* are injurious, for they are very exhausting, often render sleep broken or uncomfortable, and introduce great risk of chill. They are very erratic, come and go even in the same case, and may be entirely absent in some of the most acute cases. Still, except as an indication, they are not of great importance, for they can, as a rule, be easily controlled by drugs.

Fever.—The chief characteristic of the temperature in phthisis is its irregularity; it varies from day to day and oscillates widely in the twenty-four hours. As a rule, those cases of phthisis with the highest mean temperature are in the most active stage. The temperature does not remain for long, *i.e.*, for more than an hour or so, at its maximum, but quickly falls, it may be many degrees, thus producing the wide oscillations referred to. The smaller this drop, *i.e.*, the higher the mean level of temperature, the more acute the case. In slight cases the oscillations are much less wide, for the temperature not only does not rise so high, but it also does not fall so low. So far as the patients' feelings go, they

often say they feel better with a high temperature than with a low one. When the end is near, the temperature may run up to a great height, to 106° or 108°, or fall and remain much below normal. Either extreme is of bad omen.

The pulse.—Undue rapidity of the pulse is unfavourable as indicating great irritability of the heart or nervous system, and in this respect a fall of a few beats is of favourable significance.

The respirations may be accelerated from transient causes, *e.g.*, exertion, excitement, or secretion in the tubes, but a continued rise in the respiration rate is important as indicating rapid advance in the disease, and the existence probably of more extensive mischief than the physical signs show. The respiration rate may run up to 50 or 60, and if this be not due to some complication like pneumonia or bronchitis, it is of itself a bad sign. It may be the only evidence of acute miliary tuberculosis of the lung.

The physical signs are of subordinate importance in prognosis. They show only the amount of mischief done, not what is being done, and it is not easy to determine from them alone, except after a time, that which is the really important thing to know, viz., whether the disease be progressing or not. On the other hand, the physical signs give evidence of the extent to which the lungs are involved, and thus become of importance in prognosis; for other things being equal, the more extensive the disease, the more serious is the case. In this regard even slight physical signs may possess great significance. For example, in a case of apex-phthisis the presence in the lower parts of the lung of such physical signs as wheezing and crepitation, even though there be no alteration in the percussion, or in the voice- and breath-sounds, and especially if there be a difference between the two sides, will point to wide dissemination of the tubercular lesions; a suspicion which will be confirmed if the dyspnœa and cyanosis be in excess of what the physical signs account for. In such a case there will be strong presumption that the disease is widely disseminated, and on that account the prognosis will be grave.

Sputum.—Its amount is of little value in prognosis, for it may be abundant in very chronic cases, and even completely absent in very acute cases. As a rule, the presence of elastic tissue and numerous bacilli show active disintegration of the lung. In acute phthisis the bacilli are generally numerous, and occur in groups or masses, but in some of the most acute cases there may be no sputum at all, and in others the sputum may be scanty, and what there is contain few bacilli. The number of bacilli vary quite irregularly from day to day, so that of themselves they give no reliable indication of the progress the disease is making.

Hæmoptysis has no constant effect upon the course of phthisis, except it be in such amount as to greatly increase the anæmia. It often seems to do good rather than harm, and though in some cases after, and apparently because of, it, the disease becomes acute, in other cases it passes by without effect; but there is nothing at the time which enables us to forecast the result.

Fœtor may indicate gangrene of the lung, but if so it will be accompanied by grave general septic symptoms, and the patient will quickly die. It is more likely to be due to infection of the secretions in a cavity with putrefactive organisms. Except for its unpleasantness, it often produces no symptoms, and not unfrequently is but a temporary complication.

Conditions which tend to reduce the health and strength, i.e., conditions which enfeeble the body and reduce the resistance it is able to offer to the progress of the disease.

The most important of these are *affections of the digestive system*, as impairing the taking or digestion of food. Loss of appetite is common, and it sometimes amounts to absolute repugnance even to the sight of food. Closely connected with this is vomiting, which the very thought of food may sometimes excite. *Anorexia*, *vomiting*, *indigestion*, and *diarrhœa* are each of grave significance, and especially in acute phthisis. Phthisical patients often do fairly well as long as the digestion holds out, but as soon as it begins to fail they lose ground rapidly.

Cough, if severe, is serious on account of the exhaustion it causes, and because it breaks the night's rest.

Want of sleep, however produced, whether by cough, dyspnœa, pain, or simple insomnia, is grave, and means must be sought to control it.

Complications of any kind greatly aggravate the prognosis in phthisis, those which involve the respiratory organs being of especial gravity, *e.g.*, laryngitis especially if tubercular, bronchitis, pneumonia, pleurisy, empyema, or pneumothorax. Of acute fevers, the most important are measles, typhoid and influenza, for these seem sometimes to excite the disease, or at any rate to start it into fresh activity.

When phthisis develops as a complication of some other serious disease, it greatly aggravates the prognosis, and, as in diabetes, neurasthenia, and chronic alcoholism, may be the actual cause of death.

Amyloid disease is a grave though not very common complication. It occurs in cases of long duration with profuse expectoration, and being the result of these conditions, does not of itself materially affect prognosis.

Mode of onset.—The most unsatisfactory cases are those in which the onset has been insidious, and where the patient has been losing health, flesh, and strength without obvious cause. Such cases rarely do well. On the other hand, a very acute onset is not by any means always as grave as it appears to be at the time. Some of these cases, which begin as actively as an acute pneumonia, ultimately become quiescent, but the uncertainty and anxiety while the acute symptoms last are very great.

General considerations are often of some aid.

Age.—Speaking generally, we know that phthisis is more likely to run a rapid course in the young adult and in elderly persons.

Previous health.—As a general rule, the better the general health, the better the prognosis. Yet there are frequent exceptions. Galloping phthisis may develop in a person apparently previously robust, and, on the other hand, in a weakly delicate person phthisis may last for years.

The diathesis.—Persons of frail and slender build, of excitable, irritable disposition, and of nervous organisation, form worse subjects for phthisis than those of placid nature and plethoric habit.

The sanitary and social surroundings.—When these are bad, and such as are of themselves likely to cause ill-health, the liability to phthisis is greater, and its progress more acute; but, on the other hand, removal to more satisfactory conditions often produces wonderful amelioration. On this account the prognosis is so far better among the poor than the rich, for if phthisis develop among the well-to-do, who are surrounded with every comfort and live under the most favourable conditions, it argues an extremely feeble resistance to the disease, and,

pro tanto, the prognosis is worse. Unfortunately, it is not possible for long to keep the poor under the more favourable conditions, and the improvement, which occurs in the hospital or sanatorium, disappears as soon as they return home; indeed, the disease then sometimes seems to run even a more rapid course than before.

The family history and family type of disease may assist prognosis, for where there have been other cases in the family, it is often found that the peculiarities of the previous cases are repeated in the later ones.

THE TREATMENT OF PHTHISIS.

If bacilli did not obtain access to the body, there would be no phthisis. Prevention, therefore, is the first object to be aimed at. To deal with this we must understand the conditions under which the bacilli thrive, whence they are derived, how they maintain themselves outside the body, and the means by which they obtain access to the body; that is, we must know the complete life-history of the bacillus. The more we know of this, the more clearly we shall see how to deal with it. This subject has been already fully dealt with in considering the etiology of phthisis.

Prevention of Infection.

Prophylaxis.—We know that the sources of bacilli are the tissues and secretions of those animals and human beings already the victims of the disease. The means by which the bacilli gain access to the body are inhalation, inoculation, and ingestion by the mouth; lastly, we know that the bacilli may maintain themselves outside the body for long periods under conditions favourable to them; those conditions are moisture, warmth, want of sunlight, and fresh air. In the case of *man* the chief risk of infection lies in the discharges from the body; these should be carefully disinfected or destroyed. With tubercular ulcers or abscesses, the wounds should be kept constantly dressed with disinfecting applications, and the dressings when removed destroyed at once by burning. The most likely source of infection, no doubt, is the expectoration of phthisical persons; this should be dealt with in a similar way; either special handkerchiefs or rags should be used, which could be quickly burnt, or the expectoration should be received into spittoons, or into special vessels carried in the pocket, which could be easily cleansed and disinfected. They should contain some 5 per cent. solution of carbolic acid, which is strong enough after very short action to destroy the infectiveness of the bacilli completely.

The standing orders at the City of London Hospital for Diseases of the Chest are as follows:—

1. The sputum must be burnt. The spittoons in the wards will be half-filled with a saturated solution of commercial carbolic acid. They must be emptied daily into galvanised iron buckets containing a mixture of small coal and sawdust, and these buckets must be emptied at once into the furnace fires.

   The spittoons must be dipped into a saturated solution of carbolic acid, and then thoroughly cleaned before being returned to the wards.
2. The cloths used for wiping the spoons, cups, and plates used by tuberculous patients must be boiled for twenty minutes after use, and before being hung up to dry.
3. The cloths used for wiping the walls must be soaked after use in a saturated solution of carbolic acid.
4. Each patient must be supplied with a drinking vessel for his exclusive use, into which the sister will pour the doses of medicine. These vessels must be cleaned regularly after use.
5. The pails used to convey the sputum from the wards to the furnace shall, when not in use, be kept in a wooden trough containing a saturated solution of carbolic acid.

So far as animals are concerned, notwithstanding the recent statements of Koch on the subject, referred to on p. 401, we must assume, until the contrary is demonstrated, that the chief source of danger to man lies in the consumption of tubercular food, especially meat and milk.

In respect of meat, all animals slaughtered for food should be carefully inspected, and, if found to be tubercular, destroyed. This comes under the domain of State hygiene.

In respect of milk, all cow-houses and dairies ought to be placed under close supervision, unsanitary stables condemned, and all diseased animals at once slaughtered.

For the purposes of diagnosis, the test of tuberculin inoculation should be employed. These measures, again, could only be undertaken by the State, for they would involve great expense, especially at first; but in the case of phthisis, great as the trouble and expense might appear, they would ultimately prove less than the cost to the State resulting from the annual waste of life and strength consequent on the disease.

Much, however, may be done by individuals by boiling the milk consumed in the houses, and thus sterilising it. There is no ground for believing that boiling milk in any way impairs its nutritive value. The time will probably come when, in spite of the prejudice against boiled milk, it will be thought just as barbarous to drink unboiled milk as to eat uncooked flesh. Milk is most dangerous when it is consumed warm or fresh from a tubercular cow, and for the same reason tubercular mothers should not be allowed to suckle their infants, even though the breast itself appears to be healthy, for the milk may be infected even though the breast be not obviously diseased.

The great natural disinfectants are sunlight and fresh air. The bacilli retain their vitality, we know, for a long time in warm damp places where there is little sun and air; such places, for example, as cellars and underground dwellings, or close, ill-ventilated, ill-kept rooms, where the windows are rarely open and little sun enters, and where dust and dirt are rarely removed. Under such circumstances phthisis often seems to become almost an affection of locality. Therefore dwellings should be dry, airy, light, and clean, and all unsanitary abodes and dirty houses condemned.

All these prophylactic measures on a large scale, as applied to the general population, belong to the domain of State hygiene and preventive medicine, but, so far as they can be applied to a family or small group of individuals, they naturally belong to the province of a medical man.

Do what we will under the artificial conditions under which we live, especially in crowded cities, I suppose we must assume that in one way or another every individual is exposed at some time to tubercular infection. This is shown by the large number of persons, presumably healthy, in whom, after death, healed tubercular lesions are found in the body, and especially in the lungs; but it is evident also that the healthy body has great powers of resistance under ordinary conditions. It is important, therefore, if it can be avoided, not to permit the normal resistance of the body to be diminished, and in the second place to increase and strengthen it when it is presumably diminished. Under ordinary circumstances, and under favourable conditions, the normal resistance of the body is sufficient for its protection; in many cases completely, and in other cases partially, the lesions produced being localised, *i.e.*, confined *in situ*, by the healthy tissues.

All the conditions which tend to keep the body in perfect health, or to restore health when it has been impaired, will, in the nature of things, diminish the risk of

tubercular infection. Thus for the prevention of phthisis all the rules of sanitation should be enforced and carried out, and the diminished mortality of phthisis of recent years is fairly to be referred to the improved sanitary conditions of our towns, workshops, and houses. The health should be watched and carefully tended from the earliest days onwards, and especially in early childhood, for it is then in all likelihood that the seeds of phthisis are for the most part sown. Children, therefore, should be brought up in healthy homes or schools, with plenty of food, fresh air, and out-door exercise. How far this is possible among the poor, and those who must work hard for their living, is another and different question, but it is something to know what is desirable.

Especially are such precautions necessary where there is already a family predisposition to the disease, for though the strength of the family predisposition has probably been over-estimated, still it counts for something.

They are important also where the chest is badly formed and the lungs are weak, for it is a general law that malformed organs are especially liable to disease. Children, therefore, and adults who are badly built, poorly developed, and of weak or sickly constitutions, require especial care. The children should be brought up out of large towns, in the country, or at the seaside, should be liberally fed and not overworked, and live as much as possible in the fresh air. When the time has come to choose an occupation for them, one should be selected which will carry on the same regime, and they should not be confined to office work, with late and exhausting hours of labour. It is impossible to specify the lines to be followed in all cases, but the general laws of health must be strictly observed throughout life, and if the health appear to flag from any cause, steps must be taken at once to restore it. It is wonderful to see what care and common sense will do for weakly children and adults, and how many with the strongest predisposition to phthisis escape, when managed rightly.

The direction in which modern science is moving renders it probable that hereafter we shall have given us a substance either tuberculin, or some allied product, which may be used for protective inoculation, if not for all cases, at any rate for those which seem most predisposed; and it is not too much to hope that before long, even if we cannot cure phthisis by such means, we shall at any rate prevent it by conferring immunity through protective inoculation.

The methods so far discussed are those of prevention, not of cure, and therefore hardly belong, strictly speaking, to the treatment of phthisis, *i.e.*, to the treatment of tubercular disease of the lung already established. Still, the doctor is often called upon, when treating a case of phthisis in a family, to lay down rules for the rest of the family which have so far escaped, in order to prevent them from falling victims to the same disease. Prevention, if not part of the actual treatment, is, at any rate, part of the practitioner's work, and requires consideration in a practical treatise; for preventive medicine, if not the highest, is, at any rate, the most benevolent part of his work.

General Treatment.

When the bacilli have gained access to the body and established themselves in some part of it, the disease may remain local for a long time, but wherever it is, its existence in the body is a constant risk, and it would be well, if possible, to get rid of the tubercular disease by surgical means. In respect of phthisis it is important to treat the initial lesions of tubercle elsewhere, for it is an established fact that, no matter where the tubercle exists, if it become disseminated or infect other organs, the lungs hardly ever escape, and, as has been stated, there is reason to refer many of the cases of phthisis in adult life to the tubercular lesions of early life or childhood.

Thus it will be important to treat the early tubercular disease in the case of glands by scraping or excising, in the case of bones by appropriate operations; and though it is true that operations sometimes seem to start an active tuberculosis, probably by dissemination of the bacilli, still the number of cases in which such an event does not occur, and in which the patients are practically cured of their tuberculosis, is very much greater.

It has been even proposed to excise portions of the lung which have become tubercular. Lung surgery for the cure of phthisis seems to be very unpromising; first, because of the gravity of the operation and the complications to which it must necessarily lead; secondly, because of the difficulty of diagnosing, with sufficient certainty to justify the risk of operation, the early cases of phthisis in which the prospects of cure would be greatest; thirdly, because of the practical impossibility of removing all tubercular tissues by any operation whatever when the disease has become so far established as to be definitely diagnosed and localised.

In speaking of lung surgery, I am now referring to operations which have for their object the cure of tubercle by the removal of the diseased tissue from the lung, and not to other operations upon phthisical lungs for other purposes; for example, the opening and draining of chronic cavities.

In most cases of visceral tuberculosis—for example, tubercular glands in the mesentery and mediastinum, tuberculosis of the intestines as well as of the lungs—surgical treatment is out of the question, and the treatment must be of a different nature.

The progressive destruction of tissue which takes place in tuberculosis is due in part to the action of the products of the tubercle bacilli, to the absorption of which also many of the symptoms of the disease are to be referred, and in part to the secondary infection of the tubercular parts by streptococci and other pyogenic organisms, to which much of the fever, diarrhœa, and other symptoms are due. The objects of treatment will therefore be, in cases where it is impossible, as in phthisis, to remove or eliminate the tubercle bacilli from the body, (1) to neutralise or destroy, or at any rate render inert, the toxins they produce, and by appropriate means to counteract and control the symptoms they cause; in other words, to diminish the virulence of the bacilli, and to increase the natural resistance offered by the body; (2) to prevent secondary infection, or, when it has occurred, to deal with its consequences. With our present knowledge we cannot separate these lines of treatment from each other, but for general purposes we may arrange our means of treatment of phthisis into three groups:

1. Those measures which have for their object the reduction of the virulence of the bacilli;
2. Those which increase the resistance of the body; and
3. Those which deal with the various symptoms as they present themselves.

How far the complications which may arise require special treatment will be considered in its proper place.

To the first group belongs the treatment of tubercle by tuberculin, the serum of immune or immunised animals, and all the antiseptic methods which have been suggested, whether by internal administration through the mouth, subcutaneous injection, or locally by means of inhalation, and sprays, or by intra-tracheal or intra-pulmonary injection.

Under the second head come all the methods of general and special hygiene, by which the body is kept in, or restored to, good health.

The third, viz., the treatment of symptoms, though often disparaged as empiric, is not only a necessary part of treatment, as relieving the patient's distress, but is truly scientific, if it be remembered that the symptoms are mainly due to the absorption of the toxins of the tubercle-bacilli and other organisms, and that the real object of symptomatic treatment is to find the antidotes to these toxic substances by which their evil effects upon the body may be counteracted.

Useful as such a general classification of the methods of treatment may be, it is not desirable or practicable to carry it out into too great detail, and it will be more convenient to discuss the special methods of treatment without more than a general reference to the classification suggested.

Preventive Inoculation.—Tuberculin.—In 1890 Koch [1] startled the world by the announcement of a "kur," or specific treatment for phthisis. The facts stated were, that by the inoculation of a certain fluid named tuberculin, guinea-pigs could be rendered immune to tubercle, and that in man a profound effect was produced upon tubercular tissues which resulted in the destruction of the diseased parts and their healing by cicatrisation. Phthisis, it was stated, could be cured with certainty in the early stages, and improved even in advanced stages. Clinical observation, which at the time had been insufficient, has not confirmed the promises then held out.

Tuberculin is a sterilised extract of tubercle cultivation. Man was found to be extraordinarily susceptible to its action—bulk for bulk more than 1500 times as susceptible as the guinea-pig—and a tubercular person much more susceptible than a healthy individual; for while 2 c.c. of tuberculin were necessary to produce the required effect upon the guinea-pig, a healthy man required only 0·25, and a tubercular man only as much as 0·01. When it is remembered that the fluid itself is a greatly diluted extract, the extraordinary toxicity of the substance may be realised.

The healthy human being reacts either not at all, or scarcely at all, to a dose of 0·01 c.c., and the same holds good with regard to patients suffering from other diseases than tuberculosis. But the case is very different when the disease is tuberculosis; the same dose of 0·01 cubic centimetre, injected subcutaneously into the tuberculous patient, caused a severe general reaction, as well as a local one.

The general reaction consists in an attack of fever, which, generally beginning with rigors, raises the temperature above 39°, often up to 40°, and even 41° C.; this is accompanied by pain in the limbs, coughing, great fatigue, often sickness and vomiting. In several cases a slight icteric discoloration was observed, and occasionally an eruption like measles on the chest and neck. The attack usually begins four to five hours after the injection, and lasts from twelve to fifteen hours. Occasionally it begins later, and then runs its course with less intensity. The patients are very little affected by the attack, and as soon as it is over feel comparatively well, generally better than before it.

The local reaction can be best observed in cases where the tuberculous affection is visible; for instance, in cases of lupus. Here changes take place which show the specific anti-tuberculous action of the remedy to a most surprising degree. A few hours after an injection into the skin of the back, that is, in a spot far removed from the diseased spots on the face, etc., the lupus spots begin to swell and to redden, and this they generally do before the initial rigor. During the fever, swelling and redness increase, and may finally reach a high degree, so that the lupus tissue becomes brownish and necrotic in places.

After the subsidence of the fever the swelling of the lupus tissue decreases gradually, and disappears in about two or three days. The lupus spots themselves are then covered by a crust of serum, which filters outwards, and dries in the air; they change to crusts, which fall off after two or three weeks, and, sometimes after one injection only, leave a clean red cicatrix behind. Generally, however, several injections are required for the complete removal of the lupus tissue.

The remedy does not kill the tubercle bacilli but the living tuberculous tissues. It has no effect upon dead tissue, as, for instance, necrotic cheesy masses; nor has it any effect upon tissues made necrotic by the remedy itself. In such dead tissue living bacilli may still be present, and if not thrown off with the necrosed tissue, may infect the surrounding tissues anew. It thus becomes necessary to protect the living tissues from infection by continued application of the remedy. It was found that the dose, when frequently repeated, had to be increased, until a very large quantity was tolerated without reaction. This fact Koch attributed to the complete destruction of all tubercular tissue, but it is probable that more influence must be attributed to the establishment of tolerance than Koch believed in at first.

[1] *D. med. Woch.*, Nov. 3, 1890.

In phthisis the general reaction is predominant, but the local changes are the same in character; the tubercular tissue is rendered necrotic and inflammation takes place round it. The separation and discharge of the necrotic tissue is much more difficult, and reinfection much more likely. To deal with this risk repeated injections are employed in increasing doses.

To this account which Koch originally gave of the action of the remedy little has been added. Clinical observation, however, has demonstrated that the remedy has unfortunately by no means the value which Koch originally claimed for it. Even in the case of lupus, where the effect is most striking and the conditions most favourable for cure, there has been no case of complete cure, while in phthisis the results have been utterly disappointing. Even in the most successful cases the improvement has not been greater than is often seen, under various other forms of treatment, when patients are taken into hospitals and carefully tended. In many instances the disease runs its course unaffected, and in others it is aggravated.

Moreover, the use of the remedy in phthisis is not without risk.[1] Thus there may be violent inflammation set up of a broncho-pneumonic type; the separation of the necrosed tissue may be associated with hæmoptysis, and it will of course leave a larger cavity behind. Reinfection of the surrounding parts is almost certain to occur, and the disease may spread with increased rapidity. Lastly, the bacilli may gain access to the blood, in which they have been demonstrated soon after inoculation, and thus lead to rapid dissemination of tubercle and the death of the patient.

Though these risks may exist, fortunately they are not common, and in the majority of instances the disease continues its course, not materially influenced by the remedy for good or ill. For these and other reasons the treatment of phthisis by tuberculin has been practically abandoned.

Tuberculin was a crude and impure substance, and attempts were made to separate its ingredients and to discover the active substance, but without success. Koch himself used a purified extract obtained by precipitating the crude extract with absolute alcohol and re-dissolving it. This was found to have no advantage over the crude extract, and the latter was almost entirely used in the clinical investigations.

Hunter[2] succeeded in separating from the crude extract certain alkaloidal substances, which raised the temperature, and he thought that, as it was possible to obtain the effect of tuberculin without the fever, so it might be possible to obviate the inflammatory action also.

Kuhne[3] made an exhaustive analysis of tuberculin extracts obtained from cultivation media of different constitution, and though he reached the conclusion that the active substance is of the nature of a deutero-albumose, he failed to isolate it.

Koch[4] has recently introduced three modifications of tuberculin, tuberculin A., O., and R., the two latter being prepared by rubbing up in a mortar dried cultures of tubercle bacilli with distilled water and centrifugalising the mixture, the fluid obtained from the first centrifugalisation being called O., and that from the others R. It is stated that tuberculin R. has all the minimising and curative effects without the disadvantages and risks of the old tuberculin.

Recent reports[5] upon tuberculin R., however, seem to be almost uniformly unfavourable.

Disappointing as tuberculin has proved to be in respect of the treatment of tuberculosis in man, its use for the diagnosis of tuberculosis in cattle is daily extending, and cattle thus diagnosed to be tubercular prove, almost without exception, to be so when slaughtered. If this test be rigidly applied, and all animals which give the reaction slaughtered, there is hope that in time the amount of phthisis in man may be greatly reduced by the removal of two of the chief sources of infection, viz., infected milk and meat.

Nor does it seem too much to hope, perhaps, that in tuberculin, or in some substance like it, may be found ere long a vaccine which may have the same protective effect against tuberculosis that vaccination has against small-pox.

The protective influence against anthrax, tetanus, and diphtheria, of the blood, or blood serum, of animals rendered immune to these diseases, suggested the same line of experiment in the case of tuberculosis. The dog, goat, and ass were the animals selected, because they were supposed to be naturally immune to tubercle. They are, however, not immune, and the experiments showed that no protection was conferred.

The blood serum taken from dogs rendered tubercular was not any more efficacious. Nor is there any evidence so far to show that immunity to tuberculosis can be conferred by any other germ disease, although statements have been made that anthrax, erysipelas, and vaccination confer protection.

[1] Virchow in *Berliner med. Woch.*, Dec. 1890, Jan. and Feb. 1891.
[2] *Brit. Med. Jour.*, July 25, 1891.
[3] *Ztsch. f. Biol.*, xxix. 308.
[4] *Deut. med. Woch.*, 1897, p. 209.
[5] Congr. of Tuberculosis, Paris, 1898.

The idea that putrefaction could destroy the virulence of the tubercle bacillus led Cantani to propose the inhalation of culture fluids of putrefactive bacteria. The results were negative, and it has been finally shown that the tubercle bacillus and the putrefactive bacteria will grow together in the same medium.

The treatment of phthisis by subcutaneous injection of cantharidate of potash was abandoned almost at once, for it was found to cause albuminuria, hæmaturia, and cystitis ; it was dangerous and useless also.

Its introduction was based upon theoretical considerations, which are of interest ; (1) cantharidin was stated to excite serous transudation in the kidneys and lungs ; (2) the serum in tubercular patients was presumed to have antitoxic properties. It was concluded, therefore, by Liebreich that the injection of cantharidin in phthisis would cause the exudation of the serum containing the tubercular antitoxin into the tubercular tissues, and thus check the progress of the disease.

Antiseptic Treatment of Phthisis.

By this is usually understood the treatment of phthisis by remedies which, from the effects they produce in checking the growth of bacilli outside the body, are thought likely to have a similar effect when introduced within the body. The remedies are numerous, and of a very miscellaneous character ; the methods of introducing them various ; and, in all cases alike, the effects are uncertain.

The modes of administration are by the mouth, by inhalation of sprays or vapours, by intra-tracheal or intra-pulmonary injection, by subcutaneous injection, and, lastly, by introduction *per rectum*.

The most convenient method is by the mouth, the most certain by subcutaneous injection. The choice of method will naturally vary with the patient, with the clinical form of the disease, and with the nature of the remedy selected.

As to the mode of action in these various methods of administration, it is in most cases by the general action of the remedy after absorption ; in a few it is presumed to be more local, as in inhalation, or direct injection into the trachea or lung ; but even in these methods the drug is in great part absorbed, and its action is more general than local.

The difficulties in the way of applying the antiseptic theory in practice are two : first, that of using the antiseptic substances in a sufficient degree of concentration to produce their effect when introduced into the body ; secondly, that of ensuring their local action upon, and confining it to, the diseased part.

Almost every antiseptic known has been tried in one way or another, and most of them have been quickly abandoned as inert or harmful. It would be waste of space to refer to any but those few which have either been most largely employed, or found most useful.

Mercury is undoubtedly one of the most powerful antiseptic substances we possess, and, beyond its influence on syphilis, has a favourable effect on other septic affections. I have seen cases which presented all the features of malignant or ulcerative endocarditis, after resisting other methods of treatment, improve at once upon the administration of mercury, and in some cases of acute phthisis, also, I have been favourably impressed with its effects.

It may be administered in the form of Hyd. c. Cretâ, or better still, in small doses of Liq. Hyd. Perchlor. The combination with iodine, *e.g.*, Pil. Hyd. Iodidi Rubri, gr. $\frac{1}{16}$, is also useful.

Its administration must be perseveringly continued for some time, and therefore requires care and watching.

I have seen patients improve greatly in general health under its use, and I think this line of treatment deserves more attention than it has recently had. The administration of mercury and its preparations by subcutaneous injection has been pretty generally abandoned. Recently, however, the favourable reports of the subcutaneous injection of a solution of cyanide of mercury in syphilis led to its trial in phthisis, but its use proved to be attended with unpleasant symptoms and danger.

Iodine, in whatever form administered, whether as iodine, iodine water, the iodides, or iodoform, has little or no influence when administered by the mouth, though it has been freely used and in large doses. These preparations are not well borne, and, as a rule, cause so much discomfort and depression that they have to be given up.

Iodoform has had its advocates. The drug has been used in large amounts, even up to 30 grains a day, and for as long as a month continuously. It is, however, not well tolerated, and produces iodism early. It may be given in the form of a pill, *e.g.*, iodoform, gr. 2 ; sugar of milk, gr. 1 ; glycerine of tragacanth, *q.s.*

Salicylic Acid, salicylate of soda, salol, and salicin, benzoic acid, and the benzoates have been largely tried, but have proved inefficacious.

Tar Preparations and their Derivates.—*Tar water* and *tar pills* are old-fashioned remedies, and the various tar-derivatives have now entirely taken their place. Foremost among these are creasote, guaiacol, and carbolic acid.

Carbolic acid is but little used now. It is best administered in the form of a pill, *e.g.*, carbolic acid, gr. ii ; glycerine, ♏ ¼ ; powdered althæa, gr. iii ; or it might be given as the sulpho-carbolate of soda, gr. x to xv, in an ounce of water.

Creasote and guaiacol are at the present moment the two favourite remedies.

Creasote.—Creasote must be given continuously for some time and in full doses. It is frequently prescribed in the form of a pill or capsules, and should be then taken on a full stomach. So administered it often disturbs the digestion, impairing the appetite and leading to unpleasant repetition.

To avoid these objections, especially when large doses seemed desirable, the drug was dissolved in sterilised oil and injected *sub cutem* into the buttocks or thighs. Striking results are often produced in this way, but the pain and discomfort caused by the repeated injections were great drawbacks. It is now given by the mouth, dissolved in cod-liver oil, in the strength of 10 or 15 minims to the drachm or more of oil. In this way large doses may be taken without disturbing digestion, for long periods together, and, according to my experience, with very great benefit.

There are several kinds of creasote ; that derived from beech wood was especially advocated by Bouchard. It has the peculiarity that it contains from 6 to 9 per cent. of guaiacol, which some other creasotes, for example, that obtained from pine wood, are quite without.

Guaiacol has been preferred to creasote, because the taste and odour were more agreeable, and it was not so likely to disagree with the stomach. It may be given dissolved in spirit of wine and diluted with water, or in cod-liver oil.

The carbonate of guaiacol has been advocated as being less irritating to the stomach. It may be given in large doses, even up to 90 grains daily, 3 to 8 grains being the usual dose to commence with. It is a tasteless powder and may be taken in milk.

Sulphuretted Hydrogen and Carbonic Acid.—It has been shown that the former gas, when introduced into the rectum, is eliminated by the lung. It is upon the stated antiseptic action of these two substances after absorption that *Bergeon's* treatment is based. This consists of the introduction into the rectum of carbonic acid gas which has been passed through water saturated with hydrogen sulphide. It is an unpleasant method of treatment, and experience has not shown that it has any compensating advantages.

Formic aldehyde has been recently advocated[1] for administration by intravenous injection. The strength employed was 1 in 2000, the amount introduced being at the rate of 2 cub. centim. of the solution for each heart-beat, to give, theoretically, a strength of 1 in 100,000 in the blood of the left ventricle. The operation is not devoid of risk, it seems, and the method is only on its trial, but I fear is not promising.

Arsenic.—Arsenical cures have been often tried and abandoned. Recently the use of cacodylate of sodium has been advocated, and large doses of it given, although it contains 53 per cent. of arsenious acid. Although some patients seem to tolerate an initial dose of as much as 3–4 grains three times a day, in others the signs of arsenical poisoning are produced by much smaller doses.

As the remedy would have to be taken for a long time continuously, the treatment would involve all the risks attendant on the prolonged administration of arsenic, and cannot, therefore, be recommended.

[1] Maguire, *Lancet*, 1900, ii. p. 1712.

Inhalations, vapours, sprays.—The chief action of drugs administered in these ways is upon the upper air passages. They relieve cough and produce expectoration, but they have been largely employed with the idea that the diseased parts of the lung may be influenced by the medicaments introduced with the air. There are three objections to this theory: first, that sprays are almost entirely deposited in the throat and larynx; secondly, that if they reach the air passages at all, they do not travel far, and though portions may in time reach the lung tissue, they will be carried into the parts into which the air entry is freest, that is, into the healthy and not into the diseased parts; this objection applies to the use of vapours also; and, lastly, that it is difficult to employ substances in strength sufficient to have any bactericidal or antiseptic action at all.

When sprays are used, large quantities are, of course, swallowed, and will be absorbed from the stomach. Vapours also are freely absorbed when introduced into the air passages, so that, in estimating the effect of both sprays and vapours allowance must be made for the action of the drugs employed after absorption.

Sprays.—Solutions of benzoate and salicylate of soda, of sodium chloride, and many other substances have been freely employed in this way, but they produce no effect beyond their local action on the air-tubes, other than is produced by absorption from the stomach.

Vapours.—There is no evidence that vapours have any bactericidal action at all, and the proper place to consider their use will be under the treatment of symptoms.

Oxygen has been advocated, but whatever effect it may have upon the general condition, it has none on the disease.

Hydrofluoric acid (*i.e.*, inhalation of air passed through water containing 50 parts of the dilute acid with 150 parts of water) was in vogue for a time, but its results were either negative or deleterious.

Superheated air is a useless method, for it is shown that even when the air in the apparatus is between 200–300° F.,[1] that in the trachea is not raised more than a degree or two, while a thermometer placed between the lobes of the lung is quite unaffected.

Intra-laryngeal Injection.—To secure a larger amount of fluid passing into the air-tube than is possible by inhalations or sprays, intra-laryngeal and -tracheal injection have been employed, the fluid being injected by means of a syringe passed through the larynx direct into the trachea. There is, however, no evidence to show that the fluids injected reach the diseased parts, or have any direct action on the lung, other than can be attributed to their local action or to absorption. The procedure is less easy than spraying, and yields no better results.

This method of treatment was first suggested by Romberg[2] in 1885. It was experimentally proved that fluid in large quantities could be injected into the trachea of animals without discomfort, that it was carried into the alveoli, and that it was readily absorbed.

The quantities injected in man have varied much, generally one drachm or less, but sometimes as much as two drachms at a sitting two or three times a day, the syringe being passed through the glottis, and the injections made direct into the trachea. As a rule, it is necessary to brush the throat over with solution of cocain first.[3]

The following solutions have been used:—Menthol, 10 per cent., or benzosol, 10 per cent.; guaiacol, 2 per cent., in olive oil or in glycerine and spirit.

Intra-pulmonary Injections.—This method consists in the direct injection of antiseptic solutions into the diseased parts of the lung or their immediate neighbourhood by means of a needle introduced through the chest walls. The substances used have been various—perchloride of mercury, carbolic acid, thymol, menthol, creasote, camphor or naphthol, chloride of zinc, nitrate of silver, iodoform, iodine, iodide of soda, boracic acid, and others.

It has been shown that cicatricial tissue may be produced by these means in the lungs of healthy animals, but the nature of the tubercular process in the lungs makes it evident that the cicatrisation, even if produced, could have but little effect upon the disease. In the cases in which this method has been used in man, it has done no good, but, on the contrary, in some instances, by exciting fresh inflammation, has done positive harm.

[1] Gilmour Thompson, *New York Rec.*, 1890, No. 17.
[2] *Berl. klin. Woch.*, 1885.
[3] *Med. Chir. Trans.*, 1894.

Surgical Operations.—These are two:

1. The incision and drainage of large cavities.
2. The excision of the diseased parts of the lungs.

1. *The incision and drainage of large cavities.*—The only cases in which such a procedure would seem to be indicated are those in which there is a large chronic cavity with copious and perhaps fœtid discharge, the object being to give an external vent to the secretions, and thus remove the discomfort and risk attending expectoration through the lung.

A distinction is often drawn between chronic cavities of tubercular and those of bronchiectatic origin, operation being advocated on the latter but not on the former. But the diagnosis between them during life is, to say the least, very uncertain, for many cavities diagnosed as bronchiectatic prove to be tubercular, and a cavity of bronchiectatic origin, when it has reached a large size, has long ago ceased to be a simple bronchiectasis. The distinction has, therefore, a theoretical rather than a practical value.

Where the cavity is large, single, and at the base, it will be easy to reach and drain, and some relief may then be given if the discharge be copious; but the fistula must almost of necessity be permanent. Unfortunately, in most cases the cavity, if single, is not a simple one, but pouched and irregular. More often there are several cavities; drainage is then imperfect, and the object of the operation not achieved.

In most cases operation does no good, for practically these cavities cannot contract and heal. In many it does harm, and aggravates the discomfort which the patient suffers.

The incision of cavities at the apex, especially when of large size, is still more unfavourable in its results, and has been abandoned.

2. *Excision of the diseased parts of the lung.*—The operation has been advocated on the ground that tubercle is in its early stage, like cancer, a local disease, and that its removal would prevent its spread.

The obvious objections are that it is impossible to tell, even in the earliest cases, how far the mischief extends so as to remove all the diseased parts, and that phthisis is often but a secondary infection of the lung from some primary tubercular disease elsewhere.

A few successful cases have been reported. Thus Lowson[1] removed a portion of the right upper lobe in a woman of 34 years of age. The operation was successful, and the woman lived nine months, and died then suddenly of hæmatemesis. There was no *post-mortem* examination.

Tuffier[2] also removed the right apex in a man of 19. The wound healed completely in 120 days; the man was living and well four years later.

Against a few successful cases such as these must be set a much larger number of failures. The results of the operation are quite uncertain, and the risks very great, so that the operation is, in my opinion, not justifiable, and has been rightly abandoned.

The Treatment of Symptoms.

General symptoms must be treated on general principles. The more special symptoms of phthisis call for special consideration.

Fever.—The fever rarely requires treatment, for the temperature is seldom very high, and if high, rarely remains so, for long. Patients often feel the better with a high than a low temperature, the fever acting, no doubt, as a stimulant, the want of which the patient feels when it is absent. For these reasons antipyretics, which are very transient in their action, do not now find much favour in the treatment of phthisis. To produce any effect upon the temperature, they must be given in large doses, and, when so given, often produce unpleasant symptoms. They cannot be often repeated or long continued, so that their use must be reserved for special occasions. They are all alike given in smaller doses, but then, whatever general effect they may have, their action is not antipyretic, for in such doses they do not lower temperature.

The drugs chiefly employed as antipyretics are antipyrin, antifebrin, phenacetin and quinine, but of these the first and last have been most used.

Antipyrin has been given in 10 to 15 grains three times a day, or in larger doses, even up to 90 grains in the day. Such amounts are rarely tolerated, and even smaller doses often cause discomfort and depression. This drug is hardly ever used now.

[1] *Brit. Med. Jour.*, 1893, i. 1152.

[2] Tuffier, Paris, 1897.

The same may be said of *Salicylate of soda* and *Salicylic acid.*

Quinine, again, when given as an antipyretic, must be given in doses of 15 to 30 grains in the twenty-four hours, and such doses cannot be often repeated.

Of all these drugs alike the smaller doses have no antipyretic action.

Aconite and *digitalis*, though standing in the list of antipyretics, have little, if any, effect upon temperature, and are given for other considerations in fever, the former to reduce pulse tension—but this is usually low enough in phthisis—and the latter to raise tension and stimulate the heart. The former is contra-indicated, though the latter may be useful, if the heart be flagging.

Sponging with tepid water, either of the whole body or of the arms and legs only, is the simplest and best method of reducing temperature in phthisis. This not only reduces the temperature, but adds greatly to the comfort of the patient, who often asks for its repetition. This treatment may be repeated as often as necessary with advantage.

Cold bathing and *cold douches* are rarely employed in phthisis, and are not required.

Sweating.—Sweating, though a very common symptom in phthisis, is very inconstant. It may come and go in the most irregular way. It may be absent throughout in some cases, and it is only when considerable and frequent that it calls for treatment. It is usually spoken of as night sweating, but it would be better called sleep sweating, for it occurs night and day during and after sleep, and, as a rule, only then. It is a source of risk by chill, especially when it occurs at night. The patient, therefore, should sleep in flannel, and if the clothes become very wet or damp, they should be changed, food and stimulants being at the same time given.

Sweating depends in great part upon the general condition of the patient, and is therefore relieved by any drug or treatment which improves the general health. This is the explanation of the use of iron, quinine, acids, hypophosphites, feeding, and stimulants.

Atropine is the most reliable of all antihydrotics in phthisis; it is usually given in the form of a pill at night time, $\frac{1}{100}$, $\frac{1}{75}$, or, if necessary, $\frac{1}{50}$ of a grain of the sulphate.

A third of a grain of the extract of belladonna, or 10–15 minims of the tincture, may be given with equal effect; but the atropine pill is more convenient, and rarely fails. Its administration may be continued for a long time without any increase in the dose becoming necessary.

Some persons are very susceptible to belladonna in any form, even a small dose causing so much dryness of the mouth and discomfort that it has to be given up.

Strychnine comes after atropine, but is far inferior to it.

Three to 5 minims of the Liquor Strychninæ, or 10 minims of the tincture of nux vomica, given on going to bed, will usually be sufficient.

Oxide of Zinc was the favourite remedy before atropine and strychnine came so much into vogue. Of this 2–3 grains may be given in a pill, either alone or in combination with the same amount of extract of conium.

Camphoric Acid has been much advocated recently, and is by some considered to be superior to atropine. It may be given in doses of 5–30 grains twice daily.

Other remedies, less reliable, but useful in certain cases, are many of the *mineral acids*, *e.g.*, *nitrohydrochloric*, *sulphuric*, or *acetic*, in doses of 10–15 minims of the dilute pharmacopœial preparations; *gallic* and *tannic acids*, grains 10–15; *picrotoxin*, grain $\frac{1}{60}$; *nitrate of pilocarpin*, grain $\frac{1}{12}$. (This dose of pilocarpin often produces a slight sweat within a few moments of taking it, but checks the sleep sweats.) *Coto Bark*, 5 minims in emulsion. *Pulv. Ipecac. Co.* is useful from the opium it contains.

Agaricin has been advocated recently in doses of $\frac{1}{12}$ to $\frac{1}{6}$ of a grain, increased up to 1 grain. This takes some time, six hours or so, to act. It should be combined with Dover's powder to check its laxative action.

Lastly, *iron preparations*, especially the *sulphate* and *perchloride*; *acetate of lead*; and *digitalis*. Many of these preparations are given as stimulants, and as astringents. Their action is slow, and probably indirect, through the influence they have upon the general nutrition and tone of the body.

Cough.—The treatment of cough will vary according to its character, its cause, and the effect it produces upon the patient. The cough in phthisis is essentially conservative, having for its object the removal from the air-tubes of morbid products which would be harmful if allowed to remain. As a rule, the cough is short, not very frequent or difficult, and relieves rather than distresses the patient, so that it may require little, if any, treatment. Certainly the routine administration of opiates or sedatives for cough in phthisis is quite as likely to do harm as to do good.

The cough may, however, be frequent, violent, or paroxysmal, and cause much distress; then it must be treated, but the choice of remedies is wide, and will vary with the conditions which cause it.

Unusual frequency of a cough may be due to an unusual amount of discharge, as in cases in which there is rapidly progressing breaking down of the lungs, or where there are freely secreting cavities. If the cough be less, the secretion will be longer retained in the lungs, and experience alone can show whether the frequent cough, or the retention of the secretions, will cause most discomfort to the patient.

With chronic cavities which freely secrete, the cough is, as a rule, not frequent but paroxysmal, occurring once or twice a day, especially in the morning, and continuing until a large amount of secretion is brought up and the cavity emptied. After this, there will be little cough until the cavity has again become filled. Position largely influences the cough in such cases as these; thus by lying in some particular position, as on one side, the expectoration is facilitated, and the coughing-bout shortened, while, when rest is desired some other position is assumed, in which the fluid is more easily retained within the cavity for the time.

Violent morning cough is due to the accumulation of secretion, either in cavities or in the air-tubes during the hours of sleep, and usually varies inversely with sleep. If the night has been good, the morning cough may be severe; if the night has been disturbed by cough, the morning attack will be absent or slight. The administration of a sedative at night, to check the cough and give sleep, may be followed in the morning by such a severe bout of coughing that the patient will prefer the broken night's rest they get without the sedative.

In many cases, the morning cough is not due so much to the amount of secretion as to its viscidity and the difficulty of bringing it away. This is greatly relieved by warm drinks taken on waking or rising, as, for instance, a cup of warm milk or coffee.

In some cases the cough is out of all proportion to the amount of secretion. It is either constant or occurs in violent paroxysms. When constant, even if not very violent, it is a source of great discomfort, and may keep the patient from sleep all night; when occurring in paroxysms it may become a serious complication in the case by the exhaustion it produces, and by the pain in the head and chest it is attended by. It may lead to vomiting, and interfere with the taking and digestion of food; and of course it increases the risk of hæmorrhage. For such an irritable cough some sedative, especially opium, is indicated.

In some early cases the cough is very violent from the beginning. This is attributed to irritation in the lung or in the pleura, and relief may be given by counter irritation to the skin, with iodine for example, a mustard leaf, or a blister.

In most of these cases the cough is due to irritation in the large air-tubes, notably in the larynx or at the bifurcation of the trachea, and it is usually associated with more or less catarrh.

This kind of irritable cough is best treated with inhalations, dry or moist.

The **moist inhalations** consist of steam, impregnated or not with some balsamic or stimulating aromatic substances, *e.g.*, friar's balsam, eucalyptol, menthol, camphor, etc.; or with some more sedative drug, for example, conium.

The **dry inhalations** may be equally efficacious, and are certainly more convenient. They are administered by inspiring through special respirators or glass tubes, containing sponge or cotton wool, upon which the substances employed are sprinkled.

The substances usually given are *creasote, æther, chloroform, alcohol*, and *iodine*, as in the following prescriptions:—

Iodine, gr. iii. Æther, ʒ ii. Carbolic acid, ʒ ii. Thymol or creasote, ʒ i. Rectified spirit, ad ʒ i. 10 drops on the respirator.	Creasote, Spir. of chloroform, Carbolic acid, aa. 10–20 drops on the respirator.	Eucalyptol, ʒ iii; Ol. Pini sylv., ʒ iii; Spir. chloroform; or Spir. vini rectif., ad ʒ i.

Cough Mixtures are generally necessary. They are of the sedative class, containing *opium* or *morphia, hydrocyanic acid, chloroform, bromide of potassium, and chloral*.

Some useful cough mixtures for phthisis are the following, arranged in order of strength:—

1. A few drops of paregoric on a lump of sugar;
2. An acid linctus, *e.g.*, Acet. Scillæ; Tt. Camphor Co.; Syr. Tolut., equal parts, ʒ i for the dose:
3. Liq. Morphinæ Hydrochlor., ♏ iii; Spir. chlorof., ♏ v; Glycerine and water, aa ad ʒ i;
4. Tt. Chloroformi et Morphinæ Co., ♏ x for the dose.

For the night the common prescriptions are *Pil. Ipec. e Sc., Pulv. Ipec. Co., Bromide of potassium*, and *Chloral*.

It is not good to give opium too early if other drugs will do, for as the disease is chronic and cough persistent, the opium once taken will be continued, and the danger of the opium-habit being produced is by no means small.

The paroxysms require some rapidly acting remedy, for they not only cause distress, but danger, on account of the risk of hæmoptysis.

Severe paroxysms may even require the inhalation of a little chloroform or a subcutaneous injection of morphia, but in many cases relief is given rapidly by a few minims of laudanum placed on the tongue, followed, if necessary, by a little bromide or chloral.

When the cough depends upon laryngeal disease, the remedies chosen will be determined to a great extent by the condition of the larynx, and it may be necessary to apply some sedatives, *e.g.*, cocain or morphia, directly with the brush.

The Digestion. — Other things being equal, the prognosis in phthisis depends largely upon the digestive system. Every effort must, therefore, be made to keep the digestion in good condition, by avoiding everything that may upset it, and most carefully treating any disturbance in it that may arise. This is the more necessary because the digestive powers are often feeble, partly as the result of the disease, but chiefly from the want of the accustomed air and exercise; and, once upset, there may be great difficulty in restoring them.

For this purpose the diet and general habits must be regulated. Excesses and dissipations of every kind, whether in eating, drinking, smoking, or late hours, must be avoided. Even the choice of drugs is restricted out of regard to

the digestion, so that iron, creasote, arsenic, and similar remedies are often unsuitable; and, when given, must be given with care and discrimination, and their effect upon the digestion watched.

The appetite may be ravenous, impaired, or even completely lost.

Where the appetite is ravenous, the constant yearning for food often has a real basis, and means that the body is not properly nourished, owing to defective assimilation of the food taken.

This may be controlled by regulating the meals, reducing the amount of food taken, or giving it in a more easily digestible form, *e.g.*, peptonised; or the digestion may be assisted by pepsine and acid, or acid and strychnia. This condition is not peculiar in any way to phthisis, for busy and over-worked men, living much in office, often complain more of hunger, and take food at more frequent intervals, than they do when they are in active exercise in the open air. Here the feeling of hunger varies, not with the amount of food taken into the stomach, but with the amount of it which is assimilated; when health is at a lower level less is absorbed.

Sometimes the appetite is entirely lost, and positive repugnance is felt for food.

This is purely subjective, and does not necessarily or usually depend at all upon the digestion; for the stomach can deal with all the food put into it, as is shown by the results of forcible feeding or over-feeding, such patients digesting, easily and with advantage, enormous quantities of food, such as a healthy person could hardly dispose of.

Gastric dyspepsia in phthisis produces the usual symptoms, viz., pain or fulness after food, flatulence, eructations, or vomiting.

In acute cases of phthisis it is often the direct effect of the disease; it is similar to what is met with in other conditions of acute disease with fever; and, as in them, the tongue is coated with thick or patchy fur.

A small calomel purge occasionally, with an effervescing mixture three or four times a day, little strychnia, or an acid and strychnia mixture, will do much good.

In this condition the catarrhal state of the stomach is often kept up and excited by improper food; *e.g.*, too much solid, and, especially, too much stimulant.

Nothing is much more frequent than to find these conditions due to excess of alcohol in some form or another. A little stimulant has been ordered by the doctor; one person has suggested a little rum and milk in the morning, another a glass of port-wine or egg and sherry at eleven, some whisky and water at meals, and a nightcap on going to bed, and so it happens that the patient is drinking all day long, often without being aware what amount is really being consumed every day, or what harm is being done. Some alcohol is good, but the amount must be carefully regulated.

In the less acute cases the dyspepsia is often atonic, and due in part to the anæmia, the stomach and the digestive glands being feebly nourished, like the rest of the body, and discharging their functions only imperfectly.

For this should be prescribed as much fresh air and exercise as the strength will stand, an easily assimilated preparation of iron, *e.g.*, the saccharine carbonate, some tartrate or citrate; a little strychnia; and some easily digested, or partly digested, foods.

Vomiting in phthisis has three main causes which call for different treatment:

1. It may be dyspeptic, and follow the taking of food.

This will be treated in the usual way; but the two stock and most useful remedies are effervescing alkaline mixtures with strychnia or hydrocyanic acid.

2. It may be the result of coughing, that is, the patients may cough till they vomit.

For this some sedative cough-mixture is necessary, *e.g.*, a chloromorphia draught, or a little opium.

Sometimes the food excites the paroxysms of coughing, and so, indirectly, the vomiting is the result of the taking of food, but it is not dyspeptic.

For this, too, à sedative is the best remedy, *e.g.*, a hydrocyanic draught before meals, or some bismuth mixture, combined, if need be, with a little opium.

3. The most troublesome form of vomiting is that which depends upon neither of these causes, viz., dyspepsia or cough, but upon some other, possibly toxic, cause. Extreme instances of this are fortunately rare, but vomiting may then become a very grave complication.

Arsenic and strychnia may be tried, but the cases usually require sedatives, bromides, chloral, or even morphia.

As in hysterical cases, the vomiting may be quite irregular, and, once controlled, it may be a long time before it recurs. In some cases it appears to defy almost every treatment adopted, and may, in spite of treatment, lead to death from exhaustion. As already stated, cases in which this nervous, or as it has been called reflex, vomiting, occurs are always of grave prognosis.

Irregularity of bowels, that is, constipation alternating perhaps with slight diarrhœa, is generally associated with a coated tongue and torpid digestion.

A small compound rhubarb pill, with gr. $\frac{1}{6}$ of extract of nux vomica added, and now and then a small calomel purge, will rectify this; but it usually remedies itself when the gastric digestion is restored.

Diarrhœa—The motions are usually small and frequent rather than copious. The diarrhœa may be due to the irritation of undigested food, to catarrhal conditions of the intestines, or to ulceration.

Much may be done by the regulation of the diet, so that the food should leave but little solid residue to reach the lower bowel. A small dose of castor oil, *e.g.*, a teaspoonful, with a little laudanum if necessary, given every morning, will often control it by emptying the bowel of its irritating contents.

Bismuth is useful in many cases, and may be combined with opium. A satisfactory combination is subnitrate of bismuth and Pulv. Ipecac. Co., 10 grains of each given in milk three or four times a day.

Astringents are sometimes useful, but their routine administration, without consideration of each case on its merits, is inadvisable. They are most suitable for cases in which the motions are copious.

Useful prescriptions are the following:—

Extr. Hæmatox. liq. or Tt. Catechu, or Tt. Kino, ♏ 30, Ex. Mist. Cretæ, ℥ i.

To all these draughts a few minims of Tt. Opii may be added if necessary.

In troublesome cases recourse may be had to an Enema Opii, or to some simple astringent enema, containing alum or hamamelis, the vehicle being starch or barley water.

Abdominal pain is, fortunately, not a common symptom in phthisis, but it may be very severe. Its treatment will depend upon its cause, *e.g.*, colic or peritonitis.

The ordinary treatment is usually sufficient, but in rare cases, in which abdominal pain is continued and severe, there is no resource but opium. In all cases alike, whether the pain be gastric or intestinal, rest must be insisted upon.

Stomatitis, or an *aphthous* condition of the mouth, will be relieved by a mouth wash, *e.g.*, chlorate of potash with myrrh, or by washing the mouth with water and applying glycerine and borax. The little aphthous patches or ulcers, which are often so painful, are best treated by touching them with solid nitrate of silver.

Difficulty and *pain on swallowing* are often due to chronic ulceration of the tongue, pharynx, or larynx. These affections must be treated in the usual way locally, by sedative applications, containing usually cocain or morphia.

With these affections the taking of food is sometimes the cause of so great distress that patients look forward with dread to meal-times, and sometimes refuse food altogether. In such cases great relief is given by brushing the fauces, pharynx, and all parts within reach, with a solution of cocain, a quarter of an hour or so before food is to be given.

Expectoration.—The amount of the sputum varies chiefly with the amount of bronchitis present, and will be treated accordingly.

If the secretion or breath be fœtid, antiseptic inhalations will be necessary.

Inhalations of creasote, iodine, carbolic acid, eucalyptol, friar's balsam, turpentine, or terebine are efficacious ; pills of iodoform are useful but not agreeable ; musk is a good remedy, but costly. The mouth should also be washed with a lotion of permanganate of potash, chlorate of potash, or sanitas.

Hæmoptysis.—The treatment of hæmoptysis has been already fully dealt with, and need not be further considered here.

Insomnia.—Apart from the common causes of broken rest, *e.g.*, cough or sweating, insomnia or restlessness becomes in some patients a prominent symptom and requires treatment. It is not common, except in the more severe and acute cases.

It calls for the usual remedies, viz., bromide, chloral, sulphonal, or cannabis indica ; opium and morphia are best avoided as long as possible.

Other nervous symptoms depend, as a rule, upon the general condition of the patient, and vary with it, so that they are best treated on general principles.

General Management.

In acute phthisis the patient must be treated in all ways—indeed, can only be treated—as one suffering from acute illness. The difficulties arise when the patient is not so acutely ill. If a patient with sub-acute phthisis be treated as an invalid or convalescent would be, common sense will suggest the general rules to be observed. It is obvious that such general rules cannot be applied indiscriminately to all patients alike, but will require modification to suit the peculiarities of each individual case. In general it may be said that such patients, if well enough to be about, should live regular, quiet lives, keep early hours, and avoid all dissipation and excitement ; they should be warmly clad and well fed ; they should have fresh air, exercise, and occupation as much as possible, and should avoid exposure and fatigue of either mind or body.

Fresh Air.—Although it is desirable that phthisical patients should live as much as they can in the open air, this is often not possible in the changeable climate of this country, either because of the inclemency of the weather or because the patients are too ill. It is one of the chief, and, indeed, the most important, advantages of a good climate that more out-of-door life is rendered possible.

When, from whatever reason it may be, the patient is confined within doors, the house or room occupied should be light, cheerful, warm, and, above all things, well ventilated. One great secret of keeping the house at a uniform temperature is to keep the passages warm. Where this is not done there is a constant risk of chill to patients as they pass from room to room, and cold passages are a constant danger to invalids. If the patient be confined to the bedroom, the temperature should be maintained as far as possible at about 54 to 60. A fire is good for ventilation as well as for warmth, while a sunk sash or raised sill to the window will enable the window to be raised a few inches, and enough air will pass between the sashes to keep the room fresh without draught. When the weather permits, the window should be kept open day and night.

Many of our present houses are not as well equipped in these smaller, but very important, respects as are some of the best hotels and sanatoria, especially in the frequented winter resorts.

No doubt one of the reasons why residence in these winter resorts is so beneficial is that the good of the fresh air during the day is not undone by a close and unhealthy atmosphere during the rest of the twenty-four hours, which, it must be remembered, is in the winter at least two-thirds of the whole day.

The treatment by fresh air is carried to its extreme in what is now called the open-air treatment of phthisis, which will be referred to again.

Clothing.—This should be warm but not too heavy, and suitable to the season ; for it is as great a mistake to wrap up too warmly and suffer from oppression, as it is to be too lightly clothed and suffer from cold. To avoid chill, flannel should be worn next the skin both day and night—especially at night because of the sweating.

Attention should be paid also to the feet and legs. Patients often wrap up the body well, but leave the legs too little clad or go badly shod. The feet should be protected with warm socks, cork soles, and stout boots. To keep the feet warm is one of the secrets of avoiding chill.

The question of clothing presents the greatest difficulties in women. Even when they do not wear low dresses on occasions, and this, of course, should be strictly prohibited, they usually cover too little the upper parts of the chest and lower parts of the neck. But the chief difficulty is with corsets. Nothing can be tolerated which impedes the movements of respiration, yet the corset, by confining the action of the lower ribs, throws extra work upon the upper parts of the chest, just those parts which are the seat of most mischief in phthisis. To abandon corsets involves so much change in the figure and in the fit of dresses that it is the last thing to which phthisical women willingly agree.

Bathing.—Bathing in the open should be strictly forbidden even in summer, but a tepid, or in summer if the patient be well enough, even a cold bath in the morning, followed by a good rub down with a rough towel to stimulate the circulation, is often found exhilarating and useful. A warm bath once a week or so, to keep the skin clean and in good condition, is desirable ; but from the fear of catching cold, phthisical patients often use the bath too little.

Occupation.—Many phthisical patients are well enough to work, though not fit for any kind of work, or fit to go to work every day in any weather.

The work must not entail long hours, muscular effort, fatigue, excitement, or worry ; it should not be such as to make heavy demands on the respiratory organs, for instance, speaking, reading, singing ; nor should it be in a dusty, vitiated atmosphere, such as that of factories or mills, etc. How far it is possible to meet these requirements in any given case is open to consideration.

The same considerations are important in choosing an occupation for a young person who, though not actively phthisical, is delicate and comes of a phthisical stock. For such persons an active out-of-door life is the best, even in this country, and still more in a better climate abroad.

Wasting and Anæmia, though parts of the disease itself, are aggravated no doubt by disturbed rest, coughing, anxiety, and other accidental causes. To some extent they will disappear if the disease become quiescent, and in all cases may be to some extent combated by general management, diet, and drugs.

Diet.—The diet should be liberal, as much as the digestive powers will stand, but what the exact dietary shall be will depend upon the character of the case, and the condition of the digestion.

Thus acute cases of phthisis, where there is much fever, a furred tongue, and impaired appetite, will require feeding like other acute cases chiefly on liquid food, *i.e.*, milk, beef-tea, and broth ; or if milk cannot be easily digested, whey or koumis may be substituted for it. In any case, as soon as possible the diet should be varied with some light and easily digestible solid food.

Subacute cases should be dieted like convalescents, the meals being light, easily digestible, and frequent. The usual meals of the morning, mid-day, and evening should be supplemented by light meals between, and some light food, such as beef-tea or milk, should be given at night if sleep be disturbed. It is surprising what an amount of food such patients will often take and be the better for ; nor do the digestive powers necessarily stand in constant relation with the appetite, for even when the appetite is completely lost, or when there is such actual repugnance to food that little or none is voluntarily taken, the digestive powers still remain good, and the food will be well digested if it once reach the stomach.

For such cases the treatment by *forced feeding* or stuffing was introduced. This consists in the direct introduction into the stomach, by means of an œsophageal tube, of large quantities of pounded meat or partly digested food. Debove used for this purpose a powder of dry and pounded meat, which was equivalent to about four times its weight of fresh meat. He commenced with 1 ounce in half a pint of milk, two or three times daily, and gradually

increased the amount up to 12 or 15 ounces daily, equivalent to 3 or 4 pounds of fresh meat, mixed with 5 pints of milk. The gain in weight and health was often wonderful, and as striking as in cases of neurasthenia. After a time the patients were able to take this food by the mouth, when made appetising, by flavouring it with wine or brandy, or giving it in seasoned soups.

Of course forcible feeding is only required in certain cases, but it may yield surprising and remarkable results. Forced feeding or stuffing is now often combined with open air treatment.

Massage is not, as a rule, suitable for phthisical cases, and often does harm, but it may be used sometimes to supplement the dieting and increase the appetite.

Stimulants.—The question of stimulants is always an important one, for it is sure to be raised. Alcohol in any form has no curative or remedial action on the disease. This at once disposes of the treatment of phthisis by alcohol, and it may be unhesitatingly affirmed that the systematic and deliberate administration of alcohol, to the extent of many ounces a day, is absolutely pernicious.

It has been stated that 5 or 6 ounces of brandy or other spirit, or even more, may be taken by phthisical patients continuously without harm and with advantage. I think it not unlikely that this may account for the not infrequent association of phthisis and cirrhosis of the liver, though I believe the association is less frequently seen now than it was, probably because of the recognised evils of such alcoholic treatment.

The question of stimulants is one that must be always present to the mind in estimating the condition of a phthisical patient seen for the first time. It is not rare to find patients evidently suffering from the effects of alcohol; for the furred tongue, loss of appetite, general tremulousness, with disturbed rest and evil dreams at night, indicate the cause, and all the symptoms disappear when the alcohol is stopped.

At the same time, most phthisical patients are, I believe, the better for some stimulant with their meals, for it seems to improve appetite and to assist digestion. In what special form it is best given, whether wine, spirits, or beer, must be determined by the idiosyncrasies and preferences of the patient. If alcohol does not suit it will show itself, either in impairment of the appetite or digestion, in coating of the tongue, or in uncomfortable fulness or flushing of the face after meals. With these symptoms the special stimulant must be changed, or none at all given.

The diet is often supplemented by cod-liver oil, or, when this is not well borne, by cream or good butter, extract of malt, pancreatic emulsion, glycerine, etc.

Cod-liver Oil is the most useful of all such remedies; its use is only that of a food, being an easily digestible fat, and it has no direct influence upon the disease. Some patients, and especially children, take it easily and like it, but to others, oils of any kind are nauseating.

One or two teaspoonfuls for the adult, two or three times a day, is, as a rule, sufficient, and though much larger doses are sometimes given, there appears to be no evidence that they do the patient more good than smaller doses, while they are liable to disorder digestion, and are probably only in part absorbed. In full doses the oil may have a laxative effect, and tends to keep up or increase diarrhœa when there is a tendency to it.

The oil should be given about an hour after a meal; if it cannot be tolerated then, it may be given on going to bed, so that the patient will be in a recumbent position for some time after taking it. Where the stomach tends to reject it, the dose may be preceded by an effervescing mixture containing a little hydrocyanic acid or a few grains of carbonate of bismuth.

If the oil cannot be taken pure, it may be given in milk, cold coffee, a little brandy and water, or in wine. A few drops of lemon juice before and after will take the taste away, but best of all is a small fragment of smoked herring slowly chewed.

Æther or peppermint may be prescribed with the oil, but this mixture often repeats unpleasantly.

Emulsions may be made with aromatics and potash, but they greatly increase the bulk, and are not easily made palatable.

Yolk of egg and tragacanth are two useful vehicles—as in the Emulsio Ol. Morrhuæ, B.P.C. Cod-liver oil, 8 ounces; the yolks of two eggs; tragacanth in powder, gr. 16; elixir of saccharin, ʒ i; spir. chloroform, ʒ ss; essential oil of bitter almonds, ♏ viii; distilled water, *q.s.* to ʒ xii.[1]

[1] Martindale, *Extra pharm.*, p. 299.

Many similar emulsions are made and combined with extract of malt and pancreatic emulsion.

If cod-liver oil cannot be digested at all, it may be administered by inunction; but, except in children, this is an objectionable method of employing it, and is rarely persevered in by the patient. It is then best to abandon the remedy and employ some other, *e.g.*, *cream*, *extract of malt* or *pancreatic emulsion*. All these are useful, but their efficacy falls far short of that of cod-liver oil.

Glycerine is also sometimes administered, but presents no special advantages.

Tonics and Alteratives.—*Iron* is certainly useful when it can be taken, but it may disagree with the stomach and upset the bowels.

It matters little what preparation is given, if it can be tolerated, but the milder, less astringent preparations are the safest, *e.g.*, Pulv. Ferri. Carb. Sacch., the tartrate or citrate of iron or of iron and quinine, and Blaud's pill.

Iron is not well borne in acute cases, or when the tongue is coated and the digestion feeble. In such cases, before the iron be given, the digestion must be restored by means of an acid or alkaline mixture combined with strychnia, cinchona or gentian. The strychnia has the additional advantage that it will check the night sweats also.

Arsenic and phosphorus.—Of the drugs that have a more general action upon nutrition, there are two especially which have been long in favour in phthisis, viz., arsenic and phosphorus; both, however, have the same disadvantage, that they are likely to upset the digestion.

Arsenic may be given in the form of liquor arsenicalis in an acid or alkaline mixture; or in combination with iron, as in the following pill:—arsenious acid, gr. $\frac{1}{60}$; dried sulphate of iron, gr. iii; syrup, ♏ $\frac{1}{2}$; one pill being taken three times a day after food.

Phosphorus may be given in Perles, gr. $\frac{1}{100}$, after meals; in cod-liver oil, gr. $\frac{1}{100}$ to the drachm, an unstable and unpalatable preparation; or, as a pill, either alone or in combination with iron, quinine or nux vomica.

Phosphorus in any of these forms is likely to disagree, and I have not seen advantage come from their use.

Hypophosphites, however, may be given largely and continuously, certainly without harm, and, according to many authors, with striking benefit. They seem to be good general tonics in combination with other drugs, and are rather a food than a medicine. One of the best preparations is the mixture of the hypophosphites of lime, soda, magnesium and iron, called Parrish's food.

Climate.—Climate is not a cure for phthisis, but it may help a phthisical patient to get well. There is no place on earth where phthisis cannot exist, and though, where the air is pure and the conditions of life sanitary, there will be little phthisis or none at all, still even there if the conditions of life are altered, so as to become less sanitary or even unwholesome, phthisis will develop or increase. Hence it comes that there is more phthisis in towns than in the country, and in the crowded parts of the same town than in the less crowded parts of it. Even in Australia, which was once thought to be phthisis-free, phthisis is now becoming the same scourge it is elsewhere, and especially in the large towns.

Sunlight and fresh air are potent remedies for phthisis, and those places are best for phthisical patients where they can be longest out of doors in bright sunshine and in pure air.

A good climate is a place to get well in or to convalesce in, and in order that a phthisical patient should derive full benefit from such a climate, he must be more or less convalescent, *i.e.*, getting better, or at any rate, not getting worse. In other words, the phthisis must not be in the acute or active stage.

Even in the best of climates, the patients must still be treated as invalids or convalescents, and must be taken such care of as prudence and common sense would suggest. No climate is perfect; none will do away with the need of care; yet many patients suffer by not knowing or by disregarding this, by acting as if the climate would do the impossible by running risks and doing things which at home none but the healthy and strong would do, and which an invalid should never attempt in any climate.

Going Abroad.—The question of going abroad is a very serious one. It involves the abandonment of the ordinary occupations, and, perhaps, the loss even of the means of livelihood, the breaking up of the home and family it may be, and the expenditure of much money.

What is to be aimed at is a consecutive series of good seasons, winter and summer, so that if the first winter abroad is a success, the patient may have a second and a third if necessary. This probably involves an outlay of £300 to £500 of money, two or three years of more or less enforced leisure, and a broken family life all that time. In return for this there is offered, not a certainty of cure, but only an increased chance of recovery.

On the other hand, many cases of phthisis do well in this country if carefully tended, in spite of all its disadvantages of climate. The question, then, often comes to present itself in this form—Is the increased chance of recovery worth the great sacrifices which have to be made for it?

The question, therefore, of going abroad is not one which ought to be lightly decided. On the contrary, it requires the greatest deliberation, and into it enter many considerations besides those which are purely medical. It is often a question, not of what is the best in the abstract, but of what is the best possible under the circumstances in which the patient is placed, and in every case it must be answered with especial reference to the individual concerned. It cannot be settled on general principles; for, true as such principles may be in general, they often lead to the greatest error in practice, if applied indiscriminately and without special reference to the individual case.

Patients often come to the doctor expecting him to decide offhand not only whether they should go abroad, but to what place and when; the advice is often given on insufficient knowledge of the condition and circumstances of the patient, and, as would be expected, with not infrequently lamentable results to all concerned.

Before the right conclusion can be arrived at, several preliminary questions have to be answered.

1. Is the patient in such a condition as to be likely to derive permanent benefit from a better climate than he has at home?
2. If he be fit to go; how long a journey can he undertake; and what kind of travelling will be the best?
3. What are his means? For foreign residence is largely a question of money.
4. How long can he stay away from home?
5. Who can go with him?
6. When shall he start, and how shall he go?
7. What kind of place is likely to suit him best?
8. What is the patient's general character, and how will it be affected by living in a foreign country, with houses, customs, and food all strange to him?
9. How can he occupy his time, or amuse himself?
10. What effect will separation from home and family have upon him?
11. As regards the place, must be considered—
    (*a*) How far it will meet the various requirements already specified.
    (*b*) Whether the patient is in a condition to derive full benefit from the advantages it offers.
    (*c*) The accommodation the place affords for an invalid, in respect of housing, food, and general comforts.
    (*d*) Its accessibility, not only in respect of the journey out for the patient, but for the journey home, should the patient decide to return; and especially with reference to the coming and going of friends, which does so much to make absence from home tolerable.

This is a long string of questions to be considered, but many others, too, of less importance will suggest themselves, and small as many of them may seem to be, it is just upon such trifles as these that the whole comfort of the invalid depends, and the success or failure of the sojourn abroad turns.

All these questions must be considered carefully if wise advice is to be given. The doctor must, in fact, put himself in the patient's place, and regard the question from his point of view,

taking into consideration all these personal facts in the problem, as well as the medical aspects of the case, and according as he does this, or not, will his advice be wise and beneficial, or the reverse. The most disastrous mistakes are often made by sending patients abroad without careful thought—disastrous alike to the patient, to the family, and to the doctor's own reputation.

Even with every care, the difficulties of a right decision are often enormous, but those who take most care will make fewest errors. The light and almost flippant way in which patients are sometimes advised to go abroad, with little or no regard to the special circumstances of each case, is often cruel in its results, and cannot be too strongly reprobated.

The Condition of the Patient.—If the patient is to benefit by a good climate, his condition must be such that he may be able to take full advantage of it when he gets there.

A patient, therefore, who is in the acute stage of the disease is obviously not a case to send abroad at all; nor is he either fit to travel. He is best at home, where he can be carefully nursed and tended.

Even if he be less ill than this, though with a temperature much above normal every day, and with marked constitutional signs, he is still best at home. In these respects the constitutional condition of the patient is our best guide rather than the physical signs.

If, on the other hand, the temperature is not much raised and the strength fair, so that the patient is fit to be a good deal out of doors every day when the weather permits, he may fairly be sent abroad. Of course the most suitable cases of all are those in which the constitutional symptoms are entirely absent, and the disease stationary; and this is true in any stage of the disease, whatever the physical signs may be.

The Journey.—The next question is, what kind of a journey the patient can stand.

This often limits greatly the choice of place. Some journeys are obviously out of the question on account of the exposure or fatigue they entail. If the journey be too long to be travelled right through at once, it may be broken at convenient places. In some journeys, such as that to the Riviera, the arrangements are so well made for invalids that they can travel easily right through in comfortably warmed and ventilated carriages; but even such a journey as this is tiring, and may be more than the patient can stand. If the journey be broken, the breaks must be carefully timed and thought out, so that the patient may not be exposed at night, especially in chilly or cold weather. In this country, unfortunately, all journeys abroad involve crossing the sea, and there is nothing worse for invalids than waiting on draughty piers and railway stations when the day is cold and windy, especially at night time.

In some instances a journey by ship all the way is an easier mode of travelling than by railway; still, the patient ought to be a good sailor and the weather promising. It can do no invalid good, whether a good sailor or not, to be under closed hatches for some days, in a cabin or saloon, which is poorly ventilated and perhaps occupied by other invalids too; still, in good weather and on a good vessel, travelling by steamer is one of the easiest and best modes of making a long journey.

The Means of the Patient.—This is, perhaps, the most essential question. Can the patient afford to be away from home some time without anxiety as to cost, to travel in every comfort as an invalid should, and to have every comfort at the place of destination, in respect of housing, food, amusement, and attendance?

To winter abroad as an invalid is a very expensive business, whatever be the place chosen, and often means an outlay of £300 to £500. It would often be better for a patient to be at home, surrounded by the usual comforts and occupations, than to be in a good climate without them. To be abroad, and worried by petty cares and economies, often undoes all the good the better climate might do.

Length of Stay.—It is useless to send phthisical patients away for only a month or two. They should leave in October and not return until May or June. Of course they need not stay in the same place all the time. They may, for instance, spend the autumn in Geneva or at the Italian lakes, and make their way to the Riviera as winter sets in, going up, perhaps, to the mountains as the heat of spring and summer comes on. At any rate, the whole winter must be spent away; and if the first prove a success, possibly a second and a third. All this means heavy expense, and on this score the mind must be easy, if the climate is to do good.

Companionship.—If the patient be a young man who is fairly active, he may go abroad to settle and alone; but in other cases a companion will be needed, and the question will arise, Who else can go? Preferably, of course, some member of the family, for it is difficult to find a paid person, who will at the same time be a good companion and an attentive and skilful nurse. Even from among the family it is not always easy to find a companion congenial to the patient. One of the great difficulties to be dealt with, in arranging to send a patient abroad, is the way in which it breaks up the home. If the mother be the patient, she will have to take her daughter

with her, perhaps the one who naturally would fill her place when she was away ; or if the daughter be sick and the mother have to go, she will have, in most cases, to leave her husband alone, for his profession or business makes it impossible for him to be away from home.

The character of the Invalid.—Closely connected with the question of companionship is the character of the patient. Few persons like to be sent abroad for their health. Some may enjoy it, if well enough ; but these are generally young people with no special cares or ties, to whom the novelty and change of life abroad are of absorbing interest. Most other persons look upon it as exile more or less, and to those of a despondent nature the discomforts and trials of foreign residence often make the life abroad intolerable to them.

It will also be necessary to consider what the effect upon the invalid will be of leaving home, family, and friends for so long. Many patients feel and say that they would rather live a shorter time at home, with their family and friends about them, than a longer time abroad, separated from all that makes their life worth living. To banish a phthisical mother from her family and home is often cruel, and almost as often ineffectual, for the craving for home and the grief of separation produce more harm than the climate does good. Due allowance must be made for this personal question. A compromise may be sometimes made with advantage, by choosing a place not too far away, where the mother, for instance, may feel that, if necessary, her children could always come to her, or if need be she could go to them. The idea of absolute separation is sometimes unbearable, and the fear which some patients have, lest they should die abroad, not infrequently decides them against a foreign journey.

Place.—If these preliminary questions have all been satisfactorily answered, we have next to consider what place will best fulfil the requirements, bearing in mind especially the kind of life the patients are likely to lead, whether they will be much out of doors and lead a more or less active life, or whether they are incapable of much exertion and must lead a more or less sedentary life even when out of doors.

In the latter case the place must be warm, but in the former it may be colder and more bracing.

The idiosyncrasies of the individual, again, must not be forgotten.

It will be well to ascertain what kind of place has hitherto suited best, whether a high or low place, a hot or cold place, a bracing, or a moist and more relaxing, air. Speaking generally, phthisical patients are best in a warm but not hot place, fairly high up, and with a more or less bracing air ; at any rate, great heat and great moisture do not suit many.

With respect to the place, especial consideration must be given to its accessibility and to the accommodation available. In regard of accessibility, we have to consider not only the distance of the place away and the time it takes to reach it, but also the means of travelling thither. Some places comparatively near, like Davos, are more troublesome to get to, at any rate, at certain times of the year, than places farther away, like Madeira or the Canaries.

There is further to be considered the ease with which the patient's friends and relatives can make the journey to and fro, and the facility also offered for the patient's return home if it should be necessary. Nervous patients often object strongly to the feeling of being locked up in a place from which they can hardly move, even if it were necessary, until the winter was over.

The accommodation is also an important factor. Some excellent climates are quite unavailable for the want of such hotels or dwellings, and such cooking and comforts, as an invalid must have. For these local details local knowledge is necessary, and it is well for patients to put themselves into relation with a doctor of the place as soon as they arrive, so that they may obtain the best advice as to the part of the town to live in and the actual lodgings and rooms available. A great deal turns on these points, and the information can only be supplied by a resident in the place with the necessary knowledge.

Winter Resorts.—The places recommended as winter resorts for phthisis differ so much from one another in respect of climate that it is difficult to see what they have in common, yet good results are obtained in suitable cases with all alike. Statistical comparisons are most unreliable, first because of the smallness of the numbers dealt with ; and secondly, because there is no guarantee that the cases in each group are really patients in the same stage or condition of the disease, so as to admit of fair comparison *inter se.*

The only requisite which it appears every suitable climate possesses is that it should be such as to admit of the patient being as much as possible in the open air and sunlight. Wherever phthisical patients can spend all day out of doors, and when indoors can still live in a pure atmosphere, they will do well. I have little doubt that, with the same precautions and regime that

phthisical patients voluntarily submit to abroad, they might easily live and benefit in many places at home in spite of our cold winds, moist air, and comparative want of sunlight in winter.

The time of Travelling.—If it be settled that the patient shall go abroad, he must leave the country before the bad weather sets in, that is, before November, and must time his journey so that he reaches his destination at the right season, and is not exposed on the way to great extremes of temperature.

For instance, if he were going to Australia in October, he should not go through the Suez Canal and Red Sea, but go round by the Cape; or if going to Davos, he should arrange to get there before the winter has set in.

The foreign climates fall into two groups, the warm and the cold, the latter group being represented almost entirely at present by the high Alpine valleys, such as the resorts of the Engadine and Davos.

The Alpine Resorts.—Of these Davos is best known, and what may be said of Davos applies equally to other resorts of the same kind. The places are at considerable altitude, from 4000 to 6000 feet above the sea, and are under deep snow in winter. The air is rarefied and dry, so that there is absence of mist. The sun is bright, and, by radiation from the snow, of great power, so that the warmth at mid-day may be like that of summer, although the thermometer stand far below zero. The valleys must be sheltered, so that there is no wind, for the slightest movement of the air makes the cold intense. As long as the sun is bright and the air still, the day-climate is most enjoyable. Even weakly invalids can be out of doors and sit basking in the sun, while the more active can amuse themselves with skating, tobogganing, sleighing, and other outdoor recreations. Unfortunately, at such heights, the weather is treacherous, and if the sun do not shine, the cold is intense and no invalid can venture out of doors.

Then, again, the journey thither must be made early in the year, before the snow falls. Nor can patients safely leave till the winter is over; yet they must leave then if they possibly can, to avoid the melting snows in spring, with the dampness, mist and winds they bring.

With a good winter, life in such places is pleasant for those well enough to enjoy it, but with a bad winter it is far from enjoyable.

Davos.—Davos stands 5105 feet high. It lies in a valley 10 to 15 miles in length, running from north to north-west, about half a mile broad, with mountains rising 2000 to 5000 feet above it. It is thus much shut in and protected; but it is sunny, though, from the height of the mountains round it, the day is short. The shortest day of sunshine lasts from about 10.45 a.m. to about 3 p.m., that is, about four hours. The climate is characterised by plenty of sun and little wind. Snow falls usually about November, and the most settled weather sets in about December, and lasts through January and perhaps February. In the early spring, when the snow melts, the climate becomes trying, with damp, mist, and little sun. Patients must either leave before that time, when the journey elsewhere is unpleasant, or they may have to remain within doors day after day if the spring be a bad one. The climate depends absolutely on the sun and the wind. If the sun shine and there be no wind, it is enjoyable; if not, the reverse.

Davos is a place for the fairly well—those in whom the disease is stationary, and whose vigour is sufficient to enable them to take advantage of the amusements which the place affords. These recreations, combined with the natural beauty of the Alps in winter, make the time pass pleasantly enough. Those who are less well, able only to sit out of doors, and not strong enough to bear fatigue, may profit too, but to a less degree. While those who have much fever, in whom the disease is actively progressing, who are subject to catarrh, or who suffer with complications such as laryngitis and diarrhœa, are best away, and should never have been sent at all.

When the sun goes in, the temperature falls at once, it may be 40° to 50° Fahr., so that in a moment, as it were, the climate passes from that of summer to winter. Invalids must either be indoors before sundown, and if the sun do not shine they should not go out at all: the same is true if there be any wind.

As much of the time, even on the best of days, must be spent indoors, good hotel accommodation is more absolutely essential in the Alpine winter resorts than in warmer climates. It is the personal comfort in accommodation and food that makes Davos so popular, but there are, no doubt, many other places equally suitable in respect of climate, if the accommodation were as good.

St. Moritz, **Samaden**, **Zug**, **Maloja**, though not so sheltered, have similar advantages. **Wiesen** (4770) and **Arosa** (6209), on the way from Chur to Davos, are also well spoken of, and **Andermatt**, too, from its position, might be a good place.

When leaving these places in the spring, it will be convenient to go from Davos to Montreux or Verey, and from the Engadine to Meran or Lugano.

The Riviera.—In the western Riviera, or Riviera de Ponente, where most of the winter resorts for phthisis are situated, the special characteristics of the climate are clearness of atmosphere, much bright sunshine, dryness of air, and absence of fog and mist. The temperature is not very different from that of most parts of the South of England in the winter, only 8 or 10 degs. higher on the average, but there are four to five times the number of sunny days, and less than half the number of rainy ones. What patients seek in the Riviera in the winter is rather fine weather than heat. The climate, however, is far from perfect. If the sun do not shine the temperature is low, and there is a great fall of temperature at sundown or in the shade, so that patients must be always provided with warm wraps to put on if necessary. In the sun and during the daytime it is almost too hot for a greatcoat; in the shade and at night thick wraps are necessary to keep warm. The greatest mistakes are often made by patients, who have not been warned, in going too lightly clad, and not being well provided with warm clothes.

The different towns vary chiefly in their nearness to the sea, in their height above the sea level, and in their protection from the winds of the north and north-east, which bring the cold air down from the Alps and are sharp and piercing.

The towns are all easily reached by train, and it is the facility and comfort of the journey that often determine the choice of the Riviera as a place of residence.

Hyères is rather open to the winds and is less protected than some of the other towns, but it is three miles distant from the sea. Costabelle, upon the sea, is well sheltered and in the midst of woods. In some cases this is preferable to Hyères; the salt marshes between the town and sea make the air more bracing and stimulating.

Cannes has a similar climate to Hyères. It is a much more fashionable resort; a place pleasant enough for those who can afford a villa, take their own carriage and servants, and move in the society of the place; but for persons who prefer a quiet life, some of the other resorts are preferable.

Grasse, a few miles inland, is on the southern slopes of the mountains and sheltered from the wind, gets plenty of sun, and is well provided with accommodation.

Nice is a large town, with a good deal of life, bustle, noise, and excitement. The amusements, which it provides in abundance, are those of a town, and not such as are best suited for an invalid. Nice is much exposed, and the climate is variable in consequence, and it is far inferior to other places on this coast for a winter residence for phthisical patients.

Monaco and **Monte Carlo**, the most charming spots in the Riviera, are spoiled by the gaming tables and the kind of society they bring to them. They are admirably protected, but the temptation to spend time in the gaming rooms and in the concert-halls, which ought to be spent out of doors or at home, is too great for most persons. They are not wise places to send invalids to, especially the young, for a winter, though I think, if it were not for the tables, they would become, quickly and justly, the favourite winter resorts of the whole Riviera.

Mentone offers great varieties of climate, according to the position in the place; the east bay is warm, much more shut in and lower; the west bay, and the slope of the hills along the valleys which run down to it, give endless choice of sites for residence, and many patients who cannot enjoy the proximity of the sea find themselves in a suitable climate farther up the valley. Mentone is especially a place where local advice and experience will best determine the choice of a residence or lodging. It is, I think, best for an invalid to go to the place and try one part or another, till that which suits best is discovered.

The town is well developed, with ample accommodation of all kinds, though somewhat expensive. The walks are varied, and many of them well sheltered, even when a cold wind is blowing.

Bordighera, the last town in the French part of the Riviera, stands on a promontory projecting into the sea. It is protected from the cold winds of the north, but is open to all the winds from the south and the sea. It is therefore more bracing and exhilarating, and altogether one of the most charming places on the coast. Life is also quieter and accommodation somewhat less expensive.

San Remo is another charming place. It is stated to have more sun and less rain than any of its rivals, and it is certainly less relaxing than most of them. It is bright and pleasant, but not so well provided with walks and excursions as Mentone, which, on that account, is preferred by many visitors. It is the great resort of the Germans who visit the Riviera.

Alassio is in many respects admirably fitted for a winter residence, but the accommodation is not so well developed. Invalids should, I think, be on the slopes of the hills some distance above the sea level.

Whatever place in the Riviera may be chosen as the ultimate destination, patients should leave England in October or November, stopping on their way in Geneva, or some other convenient place on the way, so as to arrive in the Riviera in December.

Egypt and the Nile.—These are the types of a warm, dry climate; the air is dry, exhilarating, and pure, the sun shines every day and all day, the sky is clear and cloudless, the temperature is high during the day, but owing to the rapid radiation falls considerably at sundown, so that the nights are cold. The climate is more reliable than the Riviera, but there are great changes in temperature during the twenty-four hours, from 70 to 75 degrees in the day to 40 degrees at night. The warmth of the day is modified by a breeze, but the occasional hot winds from the desert are very trying while they last.

Patients should arrive at Cairo about November, but should not stay there, for it is dusty and not very healthy; if they wish to be near Cairo, they might stay at Heluan les Bains, three miles away on the east bank of the Nile. December and January they should spend at Luxor, where there is good accommodation, or at the Great Pyramids.

From the middle of November to the beginning of April is the season for Egypt, but the best months for a Nile journey are January, February, and March. The north wind is cold, and, if it blow, invalids should at once get into shelter.

Patients should leave Egypt before the heat becomes too great, that is, by about the middle of April, but invalids who have spent the winter in Egypt should break the journey on the way home, say at Biarritz, Arcachon, Malaga, or the Riviera, so as not to arrive in England until June.

Algiers.—Algiers comes in respect of climate midway between the Riviera and Madeira, being less warm, moist, and relaxing than the latter, but with a smaller fall of temperature at sundown, and with less changeability of weather than the former.

There is often a good deal of dust from the desert, and the town itself is not very sanitary, but there are suburbs which are better suited for invalids, for instance, Mustapha Supérieur.

Madeira and the Canary Islands.—The climate here is warm and moist, and as Egypt is one of the driest of places, so are these islands some of the wettest. The difference in the mean temperature of summer and winter is remarkably small, under 10° Fahr.; there are no cold winds, though there are occasionally hot winds, which are very trying.

Madeira is a relaxing climate, and often does not suit on account of the languor and loss of appetite it causes. Except in Funchal the accommodation is not good, and there are difficulties in getting a level walk, as the hills rise abruptly all round the town.

The **Canary Islands** are somewhat drier, though also warmer, being farther south. At Las Palmas accommodation is good. At Teneriffe and Santa Cruz there is also a choice of altitude, and at *Oratava*, some 1200 to 2000 feet high, the accommodation is now fair.

Mogador, on the Morocco Coast, is almost in the same longitude as the Canaries, and has much the same climate, but the accommodation for invalids is stated to be defective.

The same applies to **Tangier** on the northern coast, but the accommodation here seems to be improving.

Australia.—It is no use to send a patient to Australia if he has to earn his living, and, in order to do so, must live in the large towns like Sydney and Melbourne. These have all the disadvantages of large towns in this country, and no special advantages of climate to compensate. The districts to aim at in Australia are the inland highlands of New South Wales or Queensland.

Melbourne is hot and dusty, and subject to great changes of temperature. Patients should go straight up to the Murray River, where there are now many resorts for invalids, well equipped but expensive. From Sydney invalids would be sent up to the hills, to Goulburn, Curragong, and Bathurst. In Queensland they go to the Darling Downs, or to the districts of Maranoa and Warrego.

As to the exact spot to be selected in these vast districts, local knowledge and advice must be sought; but the patients sent to any of these places must be well-to-do, for there is little chance of their earning a livelihood, and in the sanatoria the expenses are heavy. For patients who are active, arrangements may be made for rough accommodation for a very reasonable sum upon some sheep-run, where they may, as it is said, get the "run of their teeth" and a horse for a small sum of money, but the accommodation is rough, and the patients must be able to shift for themselves.

Tasmania is more bracing than Australia in summer, but colder in winter.

The neighbourhood of Hobart Town is that about which we know most as a health resort. Many patients do well in it if they are able to enjoy the climate.

New Zealand.—New Zealand offers great choice of climate, from the almost tropical climate of Auckland to the Scotch-like climate of Dunedin with frost, snow, and rain.

As a rule, the Southern Island is more bracing than the Northern. The best part is the middle of the two islands, extending from Napier to about the upper third of the Southern Island, including Wellington and Nelson. The climate here is something like that of the south of England, though the winds are keener and the air more bracing. In the Northern Island, down to Napier, the climate is moister, warmer, and relaxing.

On the whole, the neighbourhoods of Nelson and Napier are thought to be the best for phthisical patients.

The South African Highlands.—The voyage to the Cape is easy enough, but the difficulties begin usually after landing. The destination is the Highlands in the interior, and these can only be reached by a long and fatiguing journey. Invalids used generally to go as far as Port Elizabeth by steamer, whence they made their way to Grahamstown or Craddock.

Natal offers a greater choice of suitable places, which are more accessible, though entailing a rather longer sea voyage.

The war has, for the present, rendered most parts of South Africa unsuitable for invalids just now; but when it is over, and the country settles down, no doubt many good health resorts will develop and acquire a reputation, for the climate in the Highlands is dry and exhilarating; and though the summers are hot and the winters cold, they are not trying. The great objection to many parts is the violent dust-storms which often rage.

Hitherto the South African health resorts have all been difficult of access, and the accommodation not suitable for any patients except those who are fairly active, and in a condition to rough it more or less. The great objection to them all is the expense of the journey thither, and the cost of living in any comfort when the destination is reached.

The journals are full of warnings from those who know the districts well against sending cases of advanced or active disease, or invalids who are not well provided with means.

Western America.—The health resorts here fall into two groups: first, those near the Pacific Ocean in North and South California; and, secondly, those on the slopes of the Rocky Mountains.

South California has a dry marine climate, something like that of the Riviera, but it is warmer, drier, and more equable.

Los Angelos, Passadena, Sierra Madre, Santa Monica, Santa Barbara, and San Diego, are all favourite health resorts.

In *North California*, Monterey is also a favourite place. All these towns have fair, and some, really good, hotels. They are all protected from the north and east by the mountains, and are kept at a uniform and equable temperature by the black Japan current which washes the shore. This district has one great advantage, in that there is no extreme difference in temperature between the seasons of the year, so that patients can live here all the year round.

The districts of the *Rocky Mountains* may be arranged in three groups—(1) The prairies east of the Rockies, 5000 to 6000 feet high ; (2) the lower Rockies and their valleys, from 6000 to 7000 feet high, not yet much developed ; (3) the great natural parks, from 7000 to 10,000 feet high, surrounded by mountains and sheltered from the winds. The air is dry, the sun shines almost every day, at least five days out of six, the sky is bright and clear, and there is hardly any snow ; but there are great differences in temperature, and the nights are always cold, even in summer.

Near the Rockies the advantages of altitude and of warm, equable temperature are combined, but the climate is moister. September and October are the best times to come into residence, so as to grow acclimatised before the winter sets in. The cold is then often severe, but there is little snow, and that usually towards spring, when the cold winds are often very trying.

Denver, which lies 5200 feet high, is a large town and has all its disadvantages, but the accommodation is good. It stands about the altitude of Davos.

Colorado Springs is a little higher, 6023 feet above the sea, lying about as high as San Moritz. It is sheltered and dry.

Manitou Springs lies 200 feet higher still ; it is a smaller place and has less accommodation, but is quieter and more sheltered.

As in the Riviera, great changes in temperature between day and night are experienced. The best time for them all is the autumn and early winter. In spring the winds are changeable and often trying. In summer the heat is great and dust-storms are common, but patients may then go higher up the mountains or camp out.

All these districts have one advantage in common, viz., that patients may live in them all the year round, and often find professional and commercial occupation. The great disadvantage they all have alike is that they are expensive.

All the places referred to lie on the east side of the Rockies. The west side, which probably offers equal and possibly greater advantages, is not yet much known as a health resort.

In **South America** there ought to be many suitable places, and various districts have been recommended.

Certain places in the **Peruvian Andes** have been spoken well of, for instance Janja and Huancayo, near Lima (7000 to 8000 ft. in height), and Cuzco (11,250 ft.) the ancient capital of Peru.

So again the slopes of the **Cordilleras** are said to offer a good climate, and Cordova, in the Argentine Republic, has been recommended ; but it would be risky to send patients to any of these places, knowing as little as we do about them.

Sea Voyages.—The object of a sea voyage is to provide as much fresh air and light as possible without fatigue. Therefore the voyage selected must be such as will keep the patient as long as possible in a good climate, and where he will have such weather as to enable him to spend all day on deck. To be confined to the cabin or saloon all day, and day after day, is good for no one, even the strong, and is most detrimental to phthisical patients, for the ventilation is not satisfactory, crowding is often considerable, and the space is necessarily cramped.

The great objections to a sea voyage are the risk of bad weather, the risk of sea-sickness, and the difficulty of getting good food and accommodation on board.

A sea voyage nowadays is almost always made by steamer because of the better accommodation, but the increased speed shortens the journey. The objections to a sailing ship are the defective accommodation for an invalid, and the uncertainty as to the duration of the voyage.

The sea voyage usually selected is that to Australia or New Zealand round the Cape.

It should be so timed as to reach the destination in warm weather. Leaving England, as is often done, about November or December, the first few days in the Channel are often very trying, but in three or four days the temperature becomes pleasant, and continues so till the Cape is

passed. From the Cape to Australia the temperature is cooler and more bracing, and does not become hot till Australia is reached. A voyage to Australia and New Zealand and back, with a short residence there, will practically exhaust the English winter, but on no account should an invalid return to this country until May or June.

The voyage through the Mediterranean and Red Sea to India and Australia is very bad for phthisical patients on account of the heat in the Red Sea and Suez Canal. This is so trying that most phthisical patients are made worse by it, and some have been even known to die. If the journey must be made this way, the time selected must be governed by the temperature of the Red Sea, and the time chosen should be that at which the temperature there is moderate.

Two other winter voyages have many advantages, first, that to the West Indies, which lasts from six to eight weeks, but the air is rather relaxing, and not bracing enough for most patients; secondly, that to Brazil. This is the best of all, and occupies from two to three months During the greater part of the time the climate is temperate, though at times the heat may be almost tropical.

Now that the question of sea voyages has been more completely studied, with all its pros and cons, it would seem that the objections often outweigh the possible advantages, and sea voyages are not by any means as popular for phthisical patients as they were some years ago.

Winter at Home.—It is especially its dampness, changeableness, and want of sun that makes the climate of the British Isles so trying in winter, yet it is obvious that if a winter in Falkenstein is of use, a similar regime carried out in this country would also be productive of benefit. Of course the English climate is not as good as many other climates; still no climate is perfect, at any rate none within easy reach of this country, and if patients cannot go away on account of the expense or for other reasons, the best must be made of our own resorts.

The mistake generally made in these places is that patients are left too much to themselves, and, for the want of institutions where rules must be kept, they live in their own lodging or hotels, do much as fancy leads them, and are often most imprudent.

Most of the places of winter resort for chest cases lie on the sea in the south and south-west corner of England, for instance, the Isle of Wight, Bournemouth and Torquay.

Bournemouth is in the midst of pine woods, and, owing to advantages of soil, it is dry. The town itself lies in a hollow, but the best parts for residence are the higher grounds on the eastern side. Yet Bournemouth is often very cold and raw, and, if wind blows, very dusty. The best part of the town consists of villas standing in their own grounds. Sheltered walks may be obtained among the pine woods, but the life is often found very dull unless the patient has, or makes, friends.

Hastings, though lacking the pine woods, is more bracing and more lively. It is sheltered from the north and north-east, and though somewhat exposed to the east, is much less so than St. Leonards. The best parts for chest cases are those on the shore on the eastern side of the town.

Torquay is the most popular of the southern resorts. Surrounded as it is by hills which rise to 450 feet, and which protect it from the north and east, there is a wide choice of elevation for residents, the air becoming more bracing the greater the height above the sea. It is a rainy place, but fairly sunny, and often delightful when the sun shines; still it can be bleak and cold even in Torquay, and the wind must be watched. Built, as the town is, round the sloping side of a horse-shoe bay, a walk sheltered from winds and in full sunlight can almost always be found.

I am inclined to think that, on the whole, **Ventnor**, in the Isle of Wight, is the best winter climate this country can offer.

The under cliff is well sheltered from the cold winds and well exposed to the sun, while the long terraces afford plenty of space for gentle exercise.

The **West Coast of Scotland**, if it were not so damp and windy, might offer attractions, as the air is kept warm by the Gulf Stream, and snow rarely falls in any quantity, but patients are not often sent to the Scotch watering-places.

For those whose choice of climate is limited, either by want of means or because of the distance from home, the only places which have to be considered in relation with the home resorts mentioned are those of the Riviera. The journey to the south of England is short and easy, and except to the Isle of Wight, a voyage of very short duration and often perfectly calm, the crossing of the sea is avoided. The expenses are, however, considerable in either place, though somewhat less in England than in the Riviera.

The advantage seems to lie on the whole with the Riviera, because, although the expense is somewhat greater, more is obtained for the money. At the same time, if places in England were more developed as chest resorts, and institutions grew up like those in many places abroad, under medical supervision and management, and at the same time not too expensive, I feel sure they would meet the requirements of many who cannot arrange or afford to leave England, and that they would be found to be productive of almost as much benefit to the health.

If advantage can be derived in these places under the general system in vogue, there can be no doubt that equal benefit could be obtained by a similar system in many places in this country. The great difference is that patients will submit to regulations and discipline abroad, at these so-called "cures," which they will not tolerate at home. If they were equally amenable here—and why should they not be?—many patients might stay in this country who are now sent abroad.

Open-Air Treatment of Phthisis.—This method of treatment was first regularly organised by Brehmer at Göbersdorf in Silesia, and by Dettweiler in Tannus. Many similar institutions now exist in different parts of the Continent, but that which is best known and most highly developed at the present time is at Nordrach, in the Baden Black Forest.

None of these places could be recommended as good places of winter residence on account of their climate, but the system as carried out in them all has acquired much reputation, and deservedly, for certainly many patients return from their residence there greatly benefited.

The principles upon which the system rests are—

1. The maximum of fresh air and sunlight.
2. Liberal feeding—or stuffing.
3. Regulated exercise.
4. Close medical supervision.

Institutions of this kind have now been started in various parts of this country—on the Cotswold Hills, on the sea coast in many places, Bournemouth, the Isle of Wight, in Norfolk, Yorkshire, North Wales, etc. They all follow, more or less closely, the line of the institutions at Göbersdorf, Falkenstein, and Nordrach.

The following account is based upon information supplied to me by Mr. Grace Calvert, whose views are based upon considerable personal experience :—

1. **Fresh Air and Sunlight.**—The patients spend all day in the open air, in sunny spots, either in shelters or in the open, protected from the wind.

The windows of all the rooms are kept open night and day, care only being taken to avoid through draughts.

Each patient is provided with a pocket sputum-flask for day use, and a spitting cup for the night, and spitting about the grounds is strictly forbidden.

Provided that patients are constantly in the open air, there is little to be feared from cold or chills. Even new arrivals get no harm from at once adopting an open-air life.

2. Feeding.—Three large meals are given daily, with long intervals between them, during which no food is allowed.

The meals are plain but varied.

The amount varies with the individual, but 4–8 ounces of meat with vegetables, 6–10 ounces of pudding, with an ounce of butter and a pint of milk, can be taken at each meal by most patients without more than passing discomfort.

Vomiting is sometimes troublesome at first, but the stomach soon tolerates the meals if the dietary be persevered with. At the most, it may only be necessary to reduce the milk or butter for a time.

An hour's rest in the recumbent position is insisted on after each meal, and those who are not taking any exercise at all usually manage the full diet without much trouble for, at any rate, a month or two.

The increase in weight is rapid, 4–6 lbs. a week at first, but afterwards it may be only 1 lb. or so, so that in two months there may easily be a gain of 2 stones or more.

Too rapid an increase in weight is not desirable.

3. Regulated Exercise.—Exercise is not permitted till the morning temperature is not above normal. After exercise the temperature is found to be raised somewhat, but soon falls again on rest. If it rises above 100·5° after exercise, the amount of exercise is reduced.

The patients are directed to walk slowly, not more than two miles an hour, to take frequent rests, to abstain from talking, and to lie down as soon as they get back.

The clothing should be light, so as to avoid perspiration, and a mackintosh abjured entirely.

An hour's rest in the recumbent position is ordained before each meal.

4. Close Medical Supervision.—This is necessary both in respect of exercise and food. The doctor should live with the patients, personally serving out the food, so as to see that suitable meals are given to, and consumed by, them.

As the result of sanatorium treatment, the patients gain weight, the constitutional signs diminish, the night sweats rapidly cease, strength returns, and the temperature falls.

The physical signs may gradually improve, but often remain unchanged. At any rate, in many cases the disease either ceases to progress or else progresses much more slowly.

The cases which are most suitable for sanatorium treatment are the early or chronic ones, but some acute cases do remarkably well.

The difficulty is that few patients can continue the treatment long enough, and most of them return home too soon.

Sanatorium treatment cannot change the natural course of the disease; if this is expected, disappointment will follow. It will not cure the disease, but it will assist the patient to resist the inroads of it.

The sanatorium cure, like a climate cure, will take time, and, therefore, will cost money. They are but cheaper substitutes for residence abroad.

The question arises, How far these principles of treatment can be carried out at home, instead of in a sanatorium?

The objections are—

1. The difficulty of carrying out the open-air treatment in an ordinary house at all, when other members of the family have to be considered, especially in winter.
2. The want of some one at home with the authority sufficient to insist upon the feeding being persisted with and the rules observed.
3. The practical impossibility of obtaining at home the constant medical supervision which is necessary.

A modified course of sanatorium treatment might, of course, be carried out at home; and though this might be of some advantage, it would perhaps be as inferior to the full sanatorium treatment as sanatorium treatment in this country is inferior to that in a fine climate.

51. RESPIRATORY NEUROSES.

Respiratory neuroses are affections of the respiratory organs which depend upon some disturbance of the nervous mechanism without any recognised lesion to which it can be referred.

The two most important of these neuroses are **asthma** and **whooping-cough**.

Besides these there are various so-called **hysterical affections**, and lastly, those peculiar alternations of respiratory rhythm known as **periodic respiration**, of which the most important and remarkable is Cheyne-Stokes Breathing.

52. ASTHMA.

Asthma may be defined to be a paroxysm, or a series of paroxysms, of peculiar dyspnœa, usually recurrent and often periodic, commencing more or less suddenly, rapidly reaching great intensity, accompanied by loud wheezing and by defective respiratory movements both of inspiration and expiration but chiefly of the latter, with depression of the diaphragm so that the lungs are distended to the maximum, lasting a few hours and terminating sometimes less suddenly than it began, not necessarily attended with either cough or expectoration but usually ending with more or less of mucous expectoration, unattended by fever, not associated with any constant organic lesion, and compatible with good health when the attacks are past.

Description of an attack.—The patient, who may have gone to bed in perfect health, after a few hours' sleep becomes restless, and in a short time wakes up with a feeling of constriction round the chest, and a difficulty in drawing breath. He sits up in bed to breathe more easily, or moves slowly to the door or window in search of air. Soon the increasing dyspnœa makes movement impossible, and, whether standing or sitting in bed or in a chair, the patient usually leans forward upon the arms or elbows, so as to raise and fix the shoulders. Each breath drawn is laboured, slow, prolonged, and attended with a whistling, wheezing sound, often loud enough to be heard some distance off. The little air that is drawn into the lungs enters slowly, but it wheezes out still more slowly, so that expiration is much longer and more difficult than inspiration. Speech is almost impossible ; a few syllables may be gasped out at a time, but often the wants can only be made known by signs, and if there be a cough it is short and jerky like the speech.

The chest is in the condition of maximum distension, the diaphragm stands low, and there is but little respiratory movement in either ribs or diaphragm, so that though the lungs are full of air, that air is not renewed, and extreme dyspnœa is the result.

The features express the utmost distress. The forehead is covered with beads of sweat. The countenance haggard and drawn, pale at first, but, as the dyspnœa continues, becoming turgid and cyanosed.

All this time the distress is so extreme that life seems in danger. Fortunately the dyspnœa does not long continue in this extreme intensity ; the breathing gradually becomes easier, and the attack terminates usually in a few hours with the expectoration of some viscid mucus. The patient then falls asleep and wakes up exhausted but almost free from dyspnœa.

The attack being over, he can usually go about his business without much discomfort, and continues in his usual health until the next attack.

Such is a general description of the typical asthmatic paroxysm. Although in general characters all paroxysms of asthma are alike, still in minor details each case has its own peculiarities, and these peculiarities it retains, as a rule, throughout. How great room there is for variation in different cases the analysis of symptoms will show.

ANALYSIS OF SYMPTOMS.—Access.—An attack may come on quite suddenly without any warning, but often there are prodromal symptoms which are recognised by the patient as indicating its approach. Sometimes

there are experienced feelings of unusual buoyancy, liveliness and we
on the contrary, of drowsiness, depression, and indisposition for exe
other times complaint is made of odd sensations in the epigastrium,
flatulent distension, of troublesome itching, yawning or sneezing, of cramps in
the legs, or the copious passage of light-coloured urine. I have notes of one case in which each attack was preceded three hours before it began by pain under the left nipple. Whatever the premonitory symptom may be in any given case, it usually continues the same throughout, and thus serves, like the aura in epilepsy, with which it has been compared, as a warning of what is to come. It is even possible in some cases, by accepting the warning, to adopt suitable treatment and stave off the attack. A distinction, however, must be drawn between the premonitory symptoms, *i.e.*, those which precede the paroxysm, it may even be by some hours, and those other symptoms which mark the actual commencement of the attack.

Though an attack may commence at any time of the day or night, the favourite time is about midnight or in the small hours of the morning, and it often appears at the same hour with great regularity. Thus Trousseau relates that his own attacks began almost regularly as the clock struck three in the morning.

Going to bed, or taking the recumbent position especially after a late dinner or supper, often provokes an attack, and to many asthmatic patients a substantial evening meal is entirely denied for this reason. More commonly the patient goes to bed and sleeps comfortably for the first few hours of the night, and then wakes up with the fit upon him. When the patient is awake and can observe the early symptoms, the fit is noticed to begin with a sense of constriction in the throat or round the chest, a short dry cough, a tendency to wheeze, and an increased girth round the lower part of the chest, so that the clothes have to be loosened round the waist. Though at first the patient may wander restlessly about or stand at the door or window in search of air, as the attack develops movement increases the distress and soon becomes impossible.

Position.—If in bed, the patient sits with the knees drawn up, the elbows fixed, and the shoulders raised, leaning forward with the back rounded and the head resting on the hands, or sometimes with the head thrown back and lying on the pillows with which he is supported. At other times the patient gets out of bed and sits in a high-backed arm-chair with the elbows fixed on the arms of the chair, and the head resting on its back. Other patients prefer to stand up, leaning forward on a chest of drawers or the back of a chair. Whatever be the favourite position, the patient will remain in it for hours, unable to move until the paroxysm subsides. The raising of the shoulders, which is so characteristic of the asthmatic position, has for its object to fix the scapulæ, clavicles and spine, so as to give a firm purchase to the respiratory muscles which raise the ribs.

The **respirations** are long-drawn, laboured, and attended with a wheezing, whistling, stridulous sound, which may be heard a considerable distance away, even in another room. This whistling sound is peculiar, and is often one of the earliest signs of an approaching attack. It is not like the ordinary tracheal stridor, and it is still more unlike ordinary bronchitic wheezing, nor is it so harsh as the stridor of membranous croup; still it seems to be produced chiefly in the larynx, and has been referred to the glottis assuming a fixed position, and not dilating, as it normally does, on inspiration. There are, however, none of the violent movements of the larynx up and down with respiration, which occur, as in croup, where the obstruction is in the larynx.

In spite of the dyspnœa the number of respirations is not increased, and may be even considerably reduced below the normal, even as low as nine in the

...liams).[1] This slowness of respiration is due to the extraordinary ...ion of expiration which takes place, and which is perhaps the most ...kable character of the dyspnœa; but the change is even more than this, ... the whole respiratory rhythm is altered.

Thus, in health, inspiration bears to expiration, approximately, the ratio of 6 to 7; after expiration there is usually a slight pause before inspiration commences again, but between inspiration and expiration there is no pause. In asthmatic dyspnœa inspiration is short and jerky, while expiration is greatly prolonged, until the ratio between them becomes as 1 to 2, 3, or even 4. Biermer, who timed the phases in certain cases, gives the duration of inspiration as from 1 to 2 seconds, and of expiration from 4 to 5 seconds. Besides this, the post-expiratory pause is absent. In rapid breathing this pause is always absent, one phase following the other immediately, but in asthma the breathing is slower than normal, and yet the post-expiratory pause is absent, and sometimes a post-inspiratory pause occurs instead.

The two facts that strike the eye on **inspection** of the chest are its great distension and its defective movement. The whole thorax is in the condition of maximum inspiratory distension, which is perhaps a little more evident in the upper than in the lower parts, owing to the extra raising of the upper ribs.

The movements are extremely small, for though the extraordinary muscles of respiration are all in forcible action, there is little expiratory recoil, and, consequently, but little movement. What movement there is is almost entirely costal, the abdominal muscles remain rigid, and there is evidently but little action of the diaphragm. When inspiration occurs the soft parts are drawn in slightly, but the epigastrium remains prominent, and there is no such marked recession as in cases of obstruction to the main air-tubes.

The **percussion** is hyper-resonant over the whole chest, and it is evident that there is a condition of well-marked emphysema, for the cardiac dulness is absent and the hepatic dulness does not rise above the seventh rib. These facts, together with the almost entire absence of abdominal movements on inspiration, would be explained on the supposition that the diaphragm was in a condition of more or less permanent contraction.

On **auscultation**, vesicular breathing is found to be absent, and in its place is heard the wheezing which, in part at any rate, is propagated from the larynx. This wheezing, which is said by Steavenson[2] to be most marked during the second half of inspiration, exists from the very commencement of the attack. In the later stages it is accompanied by rhonchus and sibilus, and subsequently by crepitation.

Stress is often laid upon the fact that the auscultation-signs vary much from time to time, the breath sounds being completely absent for a time in particular parts, then returning in that part, and becoming absent in another. These facts are often urged in favour of a shifting spasm of the bronchi, but I do not think any other explanation is necessary than that given of similar changes in bronchitis, viz., that they are due to temporary bronchial obstruction by secretion. The physical signs may, it is stated, be even unilateral, and this has been supposed to prove the existence of unilateral spasm of the tubes. It is, however, I believe, the result either of bronchitis limited to one side or to some gross organic lesion leading to bronchial obstruction. I have not myself, at any rate, seen anything which leads me to believe that there is or can be such a thing as unilateral asthma.

The **voice** is weak and gasping, and may be toneless, not from any affection of the larynx, but from want of breath. The **vocal vibrations** and **vocal resonance** are feeble, and may be absent, but if present they are not abnormal.

[1] *Quain's Dict.* [2] *Asthma.*

A short dry **cough** often ushers in the attack, but at the height of the paroxysm it is as feeble as the voice, and does not become conspicuous until the paroxysm is subsiding and expectoration commencing.

The Sputum.—The amount of secretion is small, and is absent at the commencement of the attack; some attacks even end without any expectoration at all (asthma siccum). Where asthma is associated with bronchitis, the sputum is of the kind usual in bronchitis, but in simple asthma the sputum is characteristic. It consists of small pearly globules or tags the size of a hemp seed, resembling tapioca. The globules consist of mucus with degenerated epithelial cells (some eosinophile), and not infrequently peculiar crystals and Curschmann's spirals. It is not frothy, nor does it contain muco-pus unless bronchitis be present.

The *crystals* are small colourless octohedra, varying in size, of uncertain nature and composition, but probably composed of a crystalline mucin-like substance. As they are found only in the centre of the pearly globules, and associated with degenerate cells, it is probable that they are the product of some chemical metamorphosis of the cells. Opinions vary as to their constancy, but Ungar[1] states that he has found them in 39 consecutive cases, though in some only after careful searching. The fact that they have been observed in other diseases of an entirely different character, *e.g.*, in the sputum of chronic bronchitis, plastic bronchitis, in the blood in leukæmia, and in the semen, robs them of any specific importance, though Leyden regarded the irritation set up by them in the bronchi as the cause of the spasm, to which he attributed the attack.

Fig. 138.
Leyden's crystals, showing their relative size compared with pus-cells.

Crystals of oxalate of lime have also been found, but they are clearly merely accidents.

Curschmann's spirals.—The thin glairy translucent mucus which is expectorated in the early stage of an asthmatic attack often has a spiral arrangement. It is twisted more or less tightly, and includes cells derived from the small bronchi, in all stages of fatty degeneration. This twisting is probably due to a rotatory action of the cilia lining the small bronchi. At any rate this kind of twisting is not peculiar to asthma. The true spirals are found in the centre of these coils and consist of a thin, clear, translucent filament, probably composed of transformed mucin. The spirals are only found in the early stage of an asthmatic attack, and disappear when the sputum becomes more abundant and mucopurulent in character.

Curschmann attached specific value to the spirals, and described asthma as a peculiar and special form of inflammation of the smaller bronchioles, "bronchiolitis exsudativa."

Fig. 139.
Curschmann's spirals, from his article in *D. Arch. f. kl. Med.*, vol. xxxii.

Hæmoptysis.—Streaks of blood upon the sputum are not at all uncommon in severe, though rare in slight paroxysms, and are due to the bronchitis. Free hæmoptysis is very rare, and must, I think, in all cases be referred to some organic lesion in the lung.

In the air expired the oxygen is almost entirely replaced by carbonic acid, the nitrogen reaching from 89 to 93 per cent.

The Circulation.—It is only at the height of the paroxysm that any effect is produced upon the heart. It may then intermit or become irregular. Its sounds are always weak, owing to the emphysema. The pulse is usually slightly accelerated, but when the dyspnœa is extreme it may be rapid, weak, small and irregular.

Temperature.—There is no rise of temperature during the attack, nor after it, unless some inflammatory complication be present.

[1] *Arch. f. klin. Med.*, xxi. 4, p. 455.

Bamberger described a case in which after a severe attack the patient became unconscious for a time. In similar cases spasmodic movements of the limbs and face, and even opisthotonos, have been observed. Nervous symptoms of this kind are extremly rare, and probably depend upon the asphyxia only.

General Condition.—During the paroxysm the patient's condition is one of extreme distress, the evidence of which is manifest upon the countenance. The face has an agonised expression, the features are drawn, the complexion is pale and earthy, the forehead covered with sweat, though the body and extremities are cold. The eye is staring and congested, the aspect is sometimes almost that of a dying man. At the acme the face is turgid, the veins distended, and the complexion somewhat cyanotic, though not to the extent which the dyspnœa would suggest, and extreme cyanosis is uncommon. The mental condition, though one of great distress, is clear.

Patients who have long suffered from asthma acquire an almost characteristic conformation; emaciated, with cold thin hands, round back and high shoulders, and the body bent slightly forwards, furrowed cheeks, an earthy, somewhat dusky, complexion, eyeballs sunken in their sockets, but tumid and watery, the chest misshapen, the upper parts dilated, the lower constricted, rigid, and without pliancy, and from it hang the arns suspended, inclined backwards, and bent at the elbows.

Duration.—The duration of a paroxysm varies from one to many hours, but it is rarely less than two to three hours. Thus it may commence about one or two o'clock in the morning, last till four or five, and then permit of several hours' sleep before the patient has to rise the next day.

An attack of asthma rarely consists of a single paroxysm, but usually of a series of paroxysms, so that it may be prolonged over days or even weeks; the first paroxysm may be the most severe of the series, but more often the early attacks are mild and gradually increase in severity; then, having reached their acme, they gradually decrease until they cease altogether.

A paroxysm often ends as it began, abruptly, but more often the end is less sudden than the beginning, and this depends much upon the duration of the paroxysm; the longer ending less abruptly than the shorter, chiefly in consequence of the greater amount of bronchial exudation which accompanies it.

The termination is often marked, especially when abrupt, by eructations, the passage of flatus, or of copious high-coloured urine depositing a sediment, the urine then differing from the copious pale urine with which the attack may commence. Epistaxis has been described as an end-phenomenon by Jaccoud, but it is rare.

When the paroxysm is past, if it has been short, the breathing may be perfectly natural and the patient feel quite well, but, as a rule, the chest feels stuffy and the breath short, especially upon exertion, even for a day or two, and this is the more likely to be the case when the paroxysm has been severe.

The attack being past, the patient is often able to do with impunity what would at other times infallibly bring on an attack; conversely, the longer the interval since the last attack the greater care is required to avoid exciting one. In these respects the asthmatic paroxysm resembles an epileptic fit.

The intervals between the attacks are like those between the paroxysms, extremely variable, but recurrence is the rule almost without an exception, and it is above all things rare for a patient to have one attack of asthma only in his whole life and no more.

In general the intervals become shorter as the disease lasts, and in many cases the attacks become more severe, but, on the other hand, it not infrequently happens that as the attacks increase in frequency they diminish in severity.

The attacks may, at first, come on only at particular times of the year, as, for instance, in the early summer; after a year or two in the autumn as well; then perhaps every month or week, and sometimes every day, but asthma diurnum is very rare, though Fagge records a case in which an asthmatic patient had not been able to sleep a single night in bed for the last twenty-five years of his life. On the other hand, where the liability is passing off the intervals become greater, until the attacks finally cease.

The attacks are sometimes remarkably periodic; this in most cases depends upon the periodical recurrence of the exciting cause, as in hay asthma, or in the monthly attacks which are related to the catamenia. A similar periodicity in the paroxysms has been already referred to.

ETIOLOGY.—Under this head must be considered first the predisposing causes, *i.e.*, the conditions under which a person is most likely to develop asthma; secondly, the exciting causes, *i.e.*, the conditions most likely to excite an attack in persons subject to asthma; and lastly, the relation in which asthma stands to other diseases.

There are no means of determining the actual **frequency** of asthma. Hospital statistics are useless for the purpose, and those of private practice are likely to be misleading. Cases of asthma occur, of course, in everyone's practice, but considering their long duration, how striking the attacks are, and how unlikely their nature is to be overlooked, the fact that spasmodic asthma is not much commoner leads to the conclusion that it may be fairly called a relatively rare disease.

THE PREDISPOSING CAUSES.—Heredity.—There can be no doubt that asthma is often hereditary, but as to the importance of heredity, opinions differ much. Lebert attaches little importance to that factor, and there is also no doubt that asthma often arises without any history of the affection in any relative. Salter's statistics show that heredity could be traced in 84 out of 217 cases, *i.e.*, in 2 out of 5, so that in the remaining 3 there was no history of inheritance. The inheritance is sometimes direct from parent to child, and may run through several generations. Sometimes it avoids the direct line and appears in some collateral branch. It may also commence and terminate in the child at the same age as in the parent. Another interesting group of cases which may be mentioned in this connection is that in which, without any asthmatic tendency in the parents, the affection develops in several of the children. The influence of heredity becomes still more accentuated when the relation of asthma to other nervous affections is considered, for example, to paroxysmal coryza and sneezing, and to hay-fever.

Sex.—If all cases of asthma be taken together *en masse*, there is no doubt that it is more common in men than in women in the proportion of about 2 to 1.

Many statistics prove this, among others those of Salter, who found that out of 153 cases 102 occurred in men and 51 in women.

The other point for which Salter contended is not so clear, viz., that in early life—for example, between 20 and 30, the time at which the nervous system is most excitable—the females exceed the males, while at a later period of life, when bronchitis and heart disease are more common, males so greatly exceed females as to make the general average much in favour of males.

Age.—In respect of age, two questions present themselves for consideration: first, the age at which the first attack most commonly occurs, and secondly, the period of life during which the largest number of cases of asthma are met with. These two questions are really distinct, but they are not infrequently confounded.

There can be no doubt that the largest number of cases are met with during the middle period of life, that is, between the ages of 30 and 50, but this is only another way of stating the fact that asthma, while it begins at varying ages in early life, is an affection of many years' duration.

The age at which the first attack occurred.—Although asthma may develop at any period of life, from infancy to extreme old age, still the first attack can often be traced back to quite the early years of life.

Thus Salter states that in no less than 31 per cent. the first attack occurred during the first ten years of life, and in 80 per cent. before the age of 40.

After 50, true spasmodic asthma rarely appears for the first time, but cases in which it develops in connection with bronchitis become fairly common; in other words, there is an increasing tendency in old people for asthma to depend upon organic disease. At the same time, asthmatics live to a good old age. The following table is given by Salter to show the ages at which the first attack developed :—

During 1st year,	11 cases	} = 31	per cent.
From 1 to 10 years,	60 ,,		
10 ,, 20 ,,	30 ,,	12·8	,,
20 ,, 30 ,,	39 ,,	17	,,
30 ,, 40 ,,	44 ,,	19	,,
40 ,, 50 ,,	24 ,,	9	,,
50 ,, 60 ,,	12 ,,	5	,,
60 ,, 70 ,,	4 ,,	1·7	,,
70 ,, 80 ,,	1 ,,	0·4	,,
	225		

Some authors assert that asthma is rare in little children. They do not say that spasmodic attacks of dyspnœa are rare in them, but they refer the attacks to other causes, *e.g.*, whooping-cough, bronchitis, emphysema, etc., or to tracheal obstruction from enlarged bronchial glands. It is true that all these affections may form the starting-point of asthma, and to the list may be added measles. It is also true that when asthma occurs in the very young it is almost certain to be associated with bronchitis and emphysema, still it must, I think, be allowed that true spasmodic asthma is not, by any means, a rare affection in the young.

Salter out of the 225 cases met with it 11 times in the first year, the four youngest occurring at the ages of 9, 14, and 28 days, and at 3 months. Politzer[1] records 2 cases at the ages of 15 and 16 months.

Instances of asthma commencing at the age of 5 years, and between 5 and 10, are common enough. The predominance of catarrh in the young often makes the diagnosis difficult, and Trousseau records a case in which he himself was misled for some time.

Even in adults it is not rare for the patient to date the asthma from an attack of measles or catarrh in early childhood.

I have lately seen two cases, one in a woman of 38, and the other in a woman of 29, in both of whom the asthma followed measles at the age of 21.

Asthma in little children often disappears after a few attacks, and it is this fact probably that has led authors to refer the attacks to some transient cause like catarrh or enlarged bronchial glands. The asthma of early life not infrequently disappears at the time of puberty, and on the whole the prognosis is best in children.

[1] *Jahrb. f. kinder. Heilk.*, N.F., iii. 4, p. 377, 1870.

It is convenient next to consider how asthmatics are affected by locality, climate, season, weather, time of day, and by their social position and occupation.

The effect of **locality** illustrates best the utter capriciousness of asthma. Some patients are well in a dry place, some in a moist, some in a high locality, some in a low, some inland and some at the seaside; sometimes only one place makes them asthmatic, sometimes there is only one place in which they are free.

I remember a young girl who could not live on account of asthma in Wapping, but was well in Whitechapel. Sometimes the patient can live in one street in the same town, but not in another, for instance, in Dover Street but not in Clarges Street, as Sir Thomas Watson records, or in the upper room of the same house but not in the lower, or even in the front room of the same floor but not in the back, as was the case with another patient of Watson's in Meurice's Hotel in Paris.

So utterly capricious is asthma in respect of locality, that it is quite impossible to foretell what place will suit any particular case; all that can be said is that, as a rule, a dry air and a high locality are less likely to suit than a low-lying place with a moist atmosphere. Perhaps the best-established fact is this, that asthmatics flourish better in towns than in the country, and especially in such smoky towns as London, Manchester, Bristol or Glasgow, and are often happiest in the most insanitary parts of them, as in a case of Walshe's, who moved with advantage from Hampstead to Seven Dials. Some even enjoy the dense pea-soup fogs which half poison the healthy. Some patients have been known to find the greatest relief in the worst parts of the Metropolitan Railway, to which they rush at once when an attack is coming on.

The same uncertainty holds also in respect of **climate, season,** and **weather**. All that can be said is, that on the whole any climate, season, or weather is bad for asthma which is likely to lead to catarrh, but it does not therefore follow that those places are best which are freest from catarrh. This is often seen in the Riviera, where asthmatics lose their bronchitis, but cannot live there on account of the increased severity of their asthma. What is good for the asthma is often bad for the bronchitis, and *vice versâ*.

On the whole, asthma is commoner in the warmer months, between May and November, than in the colder months of the rest of the year, but this depends probably less upon the temperature than upon the exciting cause being commoner in the warm than in the cold season of the year.

Time of day.—Asthmatic attacks are commoner during the night than during the day, and especially in the small hours of the morning. This is to be connected in great part with sleep, and probably with the diminished cerebral control which accompanies it. In this, asthma resembles some other forms of spasmodic affection, *e.g.*, laryngismus stridulus, angina, and the early stages of epilepsy. Sometimes the mere absence of light determines an attack, the paroxysm either not developing, or if it has already begun, rapidly subsiding when light is kindled.

Social Position.—Asthma appears to be commoner among the well-to-do than among the poor. It is of course difficult to prove this by figures, but I think the common belief is justified, and I do not see to what cause to assign this difference except to social position.

Occupation has, I believe, but little effect, except so far as it exposes the patients more to the exciting causes, such as dust or catarrh.

Asthma is stated to be commoner among those who have to use the voice much, *e.g.*, teachers, preachers, lawyers, etc., but it probably only produces more inconvenience to them, and thus drives them to seek relief more persistently.

PATHOLOGY.—The term neurosis, by which the disease is so often described, indicates that it has no definite morbid anatomy. Many lesions have been found, for example, in the lungs, heart, kidneys, etc., but none is constant. It is thus evident that the pathological lesion, if there be one, has still to be discovered. The common changes found in inveterate cases are those secondary affections of the lungs, viz., emphysema and bronchitis, which result from the disease.

THE RELATION OF ASTHMA TO OTHER DISEASES.—1. To diseases of the Lungs.—Asthma stands in no definite relation to any disease of the lungs, except that, if it be long continued, it is likely to lead to permanent emphysema and chronic bronchitis. In childhood it is often started by an attack of bronchitis or measles, and the same may happen in the adult. With acute affections of either the lung or pleura, the liability to asthma usually disappears for the time, to return when convalescence is established. Phthisical patients very rarely suffer from spasmodic asthma, and if an asthmatic patient become phthisical, an event which is by no means common, the asthma usually disappears.

This occurred in the two following cases:—(1) A man, aged 32, had suffered from asthma for four years when first seen. The attacks were typical, and no physical signs of phthisis existed. Four weeks later he spat a little blood, and eight months later had definite signs of a cavity at the right apex, the asthma having been absent for many months. (2) A female, aged 36, had been asthmatic for nine years. The attacks were preceded by pain under the right nipple and they were often followed by epistaxis. Some months later she was seen with well-marked phthisis at the left apex, but the asthma had ceased.

2. To diseases of the Heart.—More or less of permanent dyspnœa is the rule with all forms of morbus cordis, and paroxysmal attacks are by no means rare. The paroxysms may be due to sudden failure of the heart, to acute œdema of the lung, to infarct or embolism, but they differ entirely from asthmatic paroxysms. True cardiac asthma is extremely rare, perhaps the rarest of all forms; it is also the most difficult to treat, and the most doubtful of prognosis.

Cases have been described under the name of cardiac asthma in which the pulse has been very rapid, over 200 in the minute, but without excessive dyspnœa. From the accounts given, they appear to me to be not cases of asthma at all, but of the functional disease of the heart, which I have described under the title of paroxysmal hurry of the heart, and which is now embraced by the general term, Tachycardia.

3. To affections of the Stomach.—Asthma is very rarely found in connection with any organic disease of the stomach, but rather with functional derangement only, and as in most instances the paroxysm does not come on until an hour or two after a meal, it is due probably rather to the circulation in the blood of some irritating product of imperfect digestion than to any actual irritation in the stomach itself.

Henoch[1] gives two cases in children in which the attacks were immediately cut short by vomiting, in the one spontaneously, and in the other after a mechanical emetic, and he refers to another which was relieved by creasote.

Even intestinal dyspepsia may be associated with asthma, and the attack may then be relieved by free evacuation. Perhaps in this connection should be mentioned those cases which have been referred to the presence of worms (*A. verminosum*).

[1] *Berl. klin. Woch.*, xiii. 18, 1876.

4. To affections of the Kidney.—Paroxysms of dyspnœa, again, are by no means uncommon in these affections, and may be due to many causes, to failure of a hypertrophied heart, to acute or chronic œdema of the lung, or to uræmia, but true asthma is rare. In uræmia, respiratory disturbances of a paroxysmal character are frequent, sometimes with dyspnœa and sometimes without. In all cases the attacks bear a closer relationship to those which occur in diabetes than in asthma, and depend probably, like them, upon some poisonous substance circulating in the blood. At any rate, true spasmodic asthma is rare in renal disease of any kind.

We may conveniently associate with renal disease those two affections which are so commonly connected with it, viz., gout and lead-poisoning, to both of which asthma has occasionally been referred.

5. To Gout.—Trousseau [1] was the first, I believe, to draw attention to the association of asthma with gout.

He recorded a remarkable case in a boy of 5 years of age, who, after suffering from asthma for two years, developed gout in his big toe. In another case of long-standing asthma, gout developed at the age of 21, after which the asthma never returned, but the patient became a martyr to hemicrania. Trousseau also records a case in which rheumatic fever commenced the illness; this was followed by neuralgic headache, and terminated in asthma, but out of the many hundreds of cases of rheumatic fever which have passed under my review, I have no note of any instance of asthma, so that this association is also interesting on account of its rarity.

6. To Lead-poisoning.—Asthma has been described in connection with lead-poisoning.

Thus Lewy [2] records a case, and Riegel [3] mentions two others, but thinks both of them doubtful, so that if this form really exist it must be very rare.

7. To affections of the Nose.—The proof of the causal connection between asthma and affections of the nose constitutes the chief advance which has been made of recent years in our knowledge of asthma.

Nasal symptoms in connection with asthma were of course familiar to the early writers on asthma, especially in connection with that form which is now called hay-fever, and numerous instances are recorded by them in which the asthmatic attack was produced by scents, smells, emanations, dusts or powders; still it is to Voltolini [4] that the credit belongs of first clearly proving the causal connection between asthma and nasal disease. In 1872 he published cases in which asthma was associated with nasal polypi and was cured by their removal. In 1874 Haenisch records other cases in which after removal of the polypi the asthma returned with a recurrence of the local disease on several occasions and was each time cured by operation. The next step was made in 1874 by Fraenkel, who showed that asthma could be associated not only with polypi, but with any chronic nasal catarrh. A series of papers followed from this and other authors (Hartmann,[5] Bresgen,[6] Joal,[7] etc.), conclusively establishing these facts. In 1882 Hack [8] wrote his important paper, which, from the attention as well as opposition it excited, led to increased study of the subject and to a great advance of knowledge. He maintained that asthma and many other nervous affections, *e.g.*, migraine, epiphora, paroxysmal catarrh, etc., were reflex neuroses originating in local affections of the nose, and that they were associated with and depended upon a peculiar erectile turgescence of the cavernous tissue on the anterior portion of the inferior turbinated bones. Fraenkel,[9] however, showed in 1884 that the reflex neuroses could originate from any part of the mucous membrane of the nose, and that the swelling of the cavernous tissue was not a necessary condition, and this view has since been confirmed by many observers. Recently Schmiegelow has published a very complete monograph, containing not only the record of a large personal experience but a comprehensive and critical review of the whole subject.

[1] *Clin. Med. Syd. Soc.*, 1867, i. 641.
[2] *Oester. Ztsch. f. prakt. Heilk.*, xvi. 6, 1870.
[3] *Ziemssen's Handb.*, iv., ii., 1875.
[4] For references, *cf.* Fraenkel, *Berl. klin. Woch.*, xviii. 16, 1881; and Schmiegelow on *Asthma*, London, 1890.
[5] *D. med. Woch.*, 1879, 373.
[6] *Volkm. klin. Vorträge*, 1882, No. 216.
[7] *Arch. gén. de Méd.*, 1882, i. 440.
[8] *Wien med. Woch.*, 1882, v. 3.
[9] *Volkm. klin. Vortr.*, No. 242.

It is obvious in the first place that asthma is not always associated with nasal affections, and that when the two conditions concur their association may be purely accidental. It may also be assumed that asthma depends upon a peculiar condition of the nerve centres, through which the paroxysms may be excited by various peripheral irritations, of which the nasal is only one among many co-ordinate groups; and lastly, that even in those cases in which local nasal disease started it, the asthma may, if of long duration, acquire, as it were, an independent existence and continue after the nasal affection, its original cause, has been cured.

In those cases in which asthma stands apparently in close causal relation with nasal affections the local affections met with have been the following:—Polyp, Chronic Rhinitis of all kinds, including even the atrophic form, Growths on the septum or turbinated bones, Nervous catarrh, Paroxysmal Sneezing and other irritations without organic lesion.

The statistics showing the frequency with which asthma and nasal affections are associated fall into two groups; in the one a series of asthma cases are taken and the number of instances determined in which nasal affections are found; in the other a series of cases of the different nasal affections are taken and the number of instances determined in which asthma is met with.

Group 1.—Lublinski[1] found that out of 500 asthmatics 143 had pathological alterations in the nose, treatment of which cured 27 and improved 13.

Group 2.—*A.* Nasal polypi.

Baecker[2] met with asthma in 9 cases out of 310 = 2·9 per cent.
Hering ,, 7 ,, 200 = 3·5 ,,
Schmiegelow ,, 31 ,, 139 = 22 ,, ; 26 of these having true spasmodic asthma, and 5 asthmatic symptoms.

B. Chronic Rhinitis.

Fraenkel out of 32 cases met with asthma in nearly one-third.
Schmiegelow out of 514 cases found asthma in 40 = 8 per cent.; true spasmodic asthma in 34 and asthma-like symptoms in the remaining 6.

One comment upon the statistics given I cannot refrain from making. It refers to the extraordinary number of cases which appear to have come under the personal observation of the authors, from which the conclusion must be drawn, either that asthma is a much commoner disease in Germany than in this country, of which there is no other proof, or that the authors have described as spasmodic asthma many cases which would not be so described in this country, and I think these considerations vitiate the statistics given.

The figures given are sufficient to establish a probable connection as regards cause and effect between the two affections, and the results of treatment prove the connection in some cases conclusively.

a. In some cases complete and permanent cure of the asthma has followed complete cure of the nasal affection. In others the asthma has been cured for the time but has returned with a recurrence of the nasal affection and has again yielded to the appropriate local treatment

	No. of cases.	Cured.	Improved.	No result.
Lublinski,[3] . .	143	27 (=18·8 per cent.)	13 (= 9 per cent.)	...
Heymann, . .	54	29 (=55 ,,)	14 (=27 ,,)	11 (=18 per cent.)
Schmiegelow, .	56	32 (=57 ,,)	11 (=20 ,,)	7 (=12 ,,)

The following illustrative cases are interesting as being two of the earliest recorded by Voltolini and Haenisch respectively. A man aged 40 had suffered from severe attacks of asthma for four years. He was found to have numerous nasal polypi which were removed, when the asthma disappeared and did not return. The second case was in a woman aged 23, who had been the victim of asthma since 10 years of age. She was greatly relieved by the removal of several polyps from the nose. They recurred and the asthma returned several times, but each time operation relieved, and in the end, with the final cure of the polyps, the asthma was completely cured.

Besides asthma, Schmiegelow records cases in which other respiratory subjective symptoms were relieved by treatment of the nasal condition. A feeling of weight upon the chest in two cases, unpleasant pressure on the epigastrium in another. In a fourth case the treatment of

[1] *D. med. Woch.*, 1886, Nos. 23 and 24.
[2] *Ibid.*, Nos. 26 and 27.
[3] *Ibid.*, 1886, Nos. 28 and 30.

chronic rhinitis with the galvano-cautery or chromic acid produced at first a violent tickling cough, and then gave sudden relief to a feeling of shortness of breath and oppression on the chest which had been long complained of.

b. In another very interesting group of cases local treatment of the nose either aggravated the attack of asthma when it was present, or excited it during the otherwise free interval.

In Schmiegelow's series this occurred in no less than 7 cases, and in one instance followed the insufflation of a powder consisting of a mixture of nitrate of silver and starch.

c. Still more remarkable is the fact that in 2 cases Schmiegelow actually produced asthma in persons who had never had asthma. In one case, that of a woman aged 25, who suffered habitually from violent attacks of catarrh and sneezing, with profuse nasal discharge, a very severe seizure was brought on lasting for several hours by simply syringing the nose with a 1 per 1000 solution of corrosive sublimate.

In the other case, that of a man aged 28, who was under treatment for epistaxis, the most violent attacks followed the cauterisation of a part of the septum with chromic acid. The paroxysms recurred at night for several nights, until the reaction in the nose consequent on the treatment had subsided, and then they ceased.

d. In another series of cases in which, without any obvious nasal disease, there have been various nasal symptoms, such as sneezing, itching of the nose, or catarrh, the asthmatic attacks have yielded easily to the insertion into the nostril of a tampon of cocaine or menthol.

Although it is clear from what has been stated that there is more in the relation of nasal irritation to asthma than has been until recent years believed, and although most marked relief is given in some cases, still, so far as we know at present, it is impossible to recognise beforehand, with certainty, the cases in which relief will be given and those in which it will fail. Inasmuch as the cure of chronic nasal disease involves prolonged, tedious, and often somewhat distressing treatment, hesitation may well be felt before advising patients to submit to it, until ordinary methods have failed, unless the indications pointing to the nose as the source of irritation are much more evident than in most cases they really are.

These conclusions are confirmed by Sir Felix Semon's experience in an interesting resumé of the subject in the *British Medical Journal* of November 9, 1901.

This is the place in which may be conveniently considered the relation in which asthma stands to **paroxysmal sneezing**, **paroxysmal coryza**, and to **hay-fever**.

Not only, as already stated, may these affections accompany asthma, but they may alternate with it.

Hay asthma, which there is no reason to regard as a special form of asthma, except so far as it is excited by a special cause, introduces hay-fever into the group of allied affections, for in a given case, or series of cases, of hay-fever, there may be traced every degree from the simple summer catarrh to well-marked asthma; and if, in families with an asthmatic history, it can be shown that sometimes the one and sometimes the other affection develops in the descendants, the proof of the pathological connection between them seems to be complete.

Ringer[1] gives one or two family histories of this kind.

I. *Grandfather.* Asthmatic many years, died at 80.

Father.

Son, aged 35. Hay asthma from a baby, but greatly influenced by locality.

Daughter, aged 24. Hay asthma since 20.

Grandson, aged 10. Asthma since 3, associated with sneezing and itching in nose, influenced by diet.

[1] *Therapeutics*, chapter on Arsenic.

II. *Mother.* Asthmatic when young, then free for several years. Hay asthma for last ten years.

Son, aged 19. Paroxysmal sneezing and itching of nose, brought on by dust and sunlight, but only in the summer; is free in winter.	*Son*, aged 30. Asthma and bronchitis since 5, preceded by cold in head and sneezing, coryza in the daytime chiefly, asthma at night.

Ringer also quotes a case of a man aged 36, whose grandfather had asthma, who himself suffered from paroxysms of sneezing for many years, and then became asthmatic, the asthma alternating with the sneezing.

I know also of one family, in which the grandfather suffered from asthma from early life, and died at 64. The father of the family and his son suffered from attacks of paroxysmal sneezing, which often came on without cause at any time of the year, but were almost invariably excited by sudden exposure to bright sunlight in the summer.

8. Affections of the Pharynx.—The pharynx being so much less sensitive than the nose, it might be anticipated that reflex neuroses from the pharynx would be far less common.

Two cases of the kind are recorded by Porter.[1] In one, a man aged 40, asthma was associated with pharyngeal polypi, and disappeared on their removal. The polyp recurred, and with it the asthma, but the cure was finally complete. The second case he records was in connection with chronic tonsillitis.

9. Affections of the Neck and Mediastinum.—Cases of this kind in which asthmatic attacks occur are usually referred to irritation of the trunk of the pneumogastric either in the neck or within the thorax. Thus asthma might be met with in connection with goître, new-growth, or enlarged glands in the neck; and with aneurysm, mediastinal tumour, or enlarged glands within the thorax. It is often impossible to exclude the effects of pressure and to be sure that the attacks are really asthmatic at all. Eustace Smith, for example, refers asthma in children in the great majority of cases to the pressure of enlarged bronchial glands, and in this way explains the frequent improvement or cure by cod-liver oil, iron, and residence at the seaside.

In my own experience, asthma, in connection with any of the affections mentioned, either in the neck or in the thorax, is extremely rare, and although it is true that tracheal obstruction from pressure may give rise to paroxysms of dyspnœa, which may be wrongly termed asthma, still, in the majority of cases, there is sufficient difference in the character of the attack to enable the diagnosis to be correctly made and the case referred to its proper category.

10. Affections of the Skin.—There are two reasons for the interest excited by the question whether there be any relation between asthma and affections of the skin—the one because of the well-known effect of skin-irritation or skin-shock upon the respiratory centres, the other because there can be traced in many of the writings on this subject an undercurrent of belief that the asthmatic paroxysm is in these cases due to the development in the bronchi of similar pathological conditions to those observed in the skin.

Trousseau was, I believe, the first to draw attention clearly to this connection, the class of skin affections being in his opinion chiefly of a herpetic or eczematous nature. Although Hyde Salter did not actually recognise this association, still a considerable number of the cases he records (nearly 8 per cent.) yield a history of some skin eruption. It is quite of recent years that most of the cases have been described, and the most complete resumé of them up to the date of the paper is to be found in Dr. Bulkley's article in the *British Medical Journal* of 1885.

[1] *Med. Rec. New York*, 1879, ii. 345.

Out of 4000 cases of skin diseases, Bulkley[1] found asthma only in 37, *i.e.*, about 0·9 per cent., those cases being excluded in which the eruption was due to syphilis or to one of the exanthemata. Gaskoin's statistics, which yield a percentage of 7·05, are vitiated by the fact that he does not sharply distinguish between asthma and other forms of dyspnœa.

The skin affections with which asthma has been associated are in the first place urticaria and eczema, which yield by far the largest number of instances; next herpes and some of the exanthemata; in the third place, but very rarely, psoriasis, syphilis and certain drug-eruptions; and lastly a miscellaneous group, the various members of which only require to be named, acne, purpura, erythema nodosum, alopeceia areata, xeroderma, and lupus vulgaris, in most of them the association being obviously merely accidental. Considering how common skin affections are and how long asthma lasts, it is clear that a large number of asthmatics would suffer from skin affections without anything more than an accidental association existing. General statistics, therefore, will be of little value, and the only cases which would be of use in this respect are those in which the two affections stand in a manifest clinical relationship to each other, for example, where the asthma and the skin eruption appear or disappear together or alternate with one another.

Eczema.—Bulkley's statistics give a percentage of 1·33.

Bulkley met with asthma in 1·33 of his eczema cases. Trousseau stated that when asthma developed in adult life, a history of some troublesome skin affection during childhood was often obtained, and quotes a case of a lad who had suffered much from eczema as a child and became asthmatic at the age of 17. West stated that he had never known eczema in children to be extensive and long-continued without a marked liability to asthma being associated with it. In all such cases we must remember how extremely common eczema is in early life and how great the chances are in any case of asthma that a history of eczema might be obtained if it were specially enquired into.

The following are some of the cases often quoted:—

Eczema of the face in a boy of 6 years in whom asthma developed every time the eczema disappeared (Caillaut).[2]

Two cases in which eczema started at 2 months of age and was followed by asthma, in one of the cases at the age of 2½ years (Steavenson).

Eczema at 14 days, dyspnœa following each improvement of the eczema (Balfour).

A family in which all the members suffered from eczema between the first and second dentition, accompanied with asthma, both affections passing off after the completion of the second dentition (Cunningham).

Eczema and asthma in a child developing together and cured together (Thorowgood).

It will be observed that all the cases quoted occurred in children. I do not know of any case of this kind in the adult; if one was met with it would be important to bear in mind the relation of eczema to gout.

Urticaria.—Of Bulkley's cases 7 per cent. (5 out of 68) had asthma, 8 others some bronchial symptoms. I am surprised at such a percentage, for it does not accord with my experience. Out of a very large number of cases of urticaria, though complaint has frequently been made of discomfort in breathing, I have never met with a case of true asthma, and the published cases are very few. The most severe case of dyspnœa occurring with urticaria was met with in a man who was remarkably susceptible to urticaria from articles of food, whose breathing became alarmingly short, but the dyspnœa was not asthmatic in character.

Trousseau[3] records a case in which urticaria was coincident with asthma and disappeared with it.

Warner[4] describes another case in which urticaria developed with patches on the fauces. Asthma and severe vomiting occurred several times during the illness, which in the seventh week was complicated with rheumatic fever. The asthma, fever, and urticaria all disappeared together on the use of salicylate of soda, having resisted all other treatment.

A case of recurrent urticaria associated with asthma is published by Ungar, and others by Schnitzler and Fenekovy.[5]

[1] *Brit. Med. Jour.*, ii., 1885, 954.
[2] For references, *cf.* Bulkley.
[3] *Clin. Med.*
[4] *Brit. Med. Jour.*, 1885, i. 483.
[5] *Berl. klin. Woch.*, 1881, No. 48.

In one of Schnitzler's[1] cases a woman 30 years of age was attacked suddenly during her third pregnancy with sneezing, after which urticaria and asthma developed, from which she had never suffered before ; the two previous pregnancies had been perfectly normal.

The most remarkable case I know is that of Fenekovy. The patient was a woman 39 years of age, who was attacked with nasal catarrh and frequent sneezing. This lasted for one or two days, and was then followed by urticaria and an attack of asthma. When the urticaria subsided the bronchial secretion became copious and the asthma ceased. These symptoms always followed in the same order and recurred at intervals of every four or five months for eight years, interrupted only by pregnancies, when the intervals were shorter and the attacks more severe.

Herpes.—The association of asthma and herpes is mentioned by Trousseau, and later by Waldenburg, but I do not know of any conclusive case recorded.

The exanthemata.—Measles is often spoken of as the starting-point of asthma both in the child and in the adult, but it is rather with the bronchitis that accompanies that disease than with the rash that the asthma should be connected. Among Salter's cases is one in which asthma followed the disappearance of a small-pox eruption.

Erythema.—The only case I know is mentioned by Bulkley as occurring in the course of erythema nodosum.

Psoriasis.—Thorowgood records a case of hereditary asthma in a brother and sister which disappeared as psoriasis developed ; and Th. Williams[2] a case of asthma in a syphilitic patient which resisted treatment until the appearance of a specific psoriasis suggested the use of iodide of potassium, when both the rash and the asthma were cured.

In connection with the use of iodide of potassium may be mentioned a case in which that drug was freely administered without effect, until acne was produced, and then the asthma yielded.

This short review of the facts demonstrates how slight the evidence is upon which the assumed connection between asthma and affections of the skin rests. In most cases the association is obviously accidental ; in others both affections were probably the joint effects of some common cause, and even the instances in which a causal connection might be conceived to exist are so few that they throw no light upon the nature of the disease.

Affections of the Nervous System.—There are, I believe, no instances in which asthma stands in anything more than an accidental relation with any recognised disease of the spinal cord or medulla. Nor are there any cases in which asthma has been proved to depend upon any actual gross intracranial lesion ; still a sufficient number of cases have been recorded, in which asthma has been met with in various forms of cerebral affection, to make this association more than a mere coincidence.

Salter gives an account of a case of hydrocephalus in which, during the last four days of life, the child suffered from two paroxysms of dyspnœa of an asthmatic character. Graves described a similar case in a child suffering from convulsions.

Salter again describes a case in which asthma alternated with epilepsy, and was on each occasion preceded by the usual aura, the only difference being that asthma followed instead of the fit. A similar case is recorded by Lloyd and Taylor[3] as occurring in an idiot woman of the age of 31.

In a case of Trousseau's, an asthmatic patient lost his asthma after an attack of gout at the age of 21, but suffered ever after from hemicrania.

Eulenberg[4] records similar cases in which asthma alternated with hemicrania, and also with angina pectoris.

The most interesting contributions to this subject have been recently made by Savage and Conolly Norman[5] in respect of the relationship between asthma and insanity, in many instances the two affections alternating, the patient being free from asthma as long as the insanity lasted, and becoming subject to it again on returning to a right mind.

[1] *Wien med. Pr.*, 1884, 1565.
[2] *Lancet*, 1873, ii. 328.
[3] *Lancet*, June 10, 1893.
[4] *Nervenkr.*, 1871.
[5] *Insanity*, 1889 ; *Jour. of Mental Sc.*, April 1885.

Savage's cases are the following:—

1. A woman, aged 34, without any insane inheritance, had been a patient in an incurable hospital for some years with chronic asthma. She became for the second time insane and lost her asthma. She continued insane until an attack of asthma came on, and then began to recover. During convalescence she had but few attacks of asthma, but when well the asthma returned with greater violence than ever.

2. A female, aged 28, with a history of insanity on the mother's side and phthisis on the father's, became asthmatic at the time of menstruation and continued so for six years, when she became insane. During her insanity she was free from asthma, but with mental health the asthma returned. She had two subsequent attacks of insanity with the same sequence of events.

3. A female, aged 32, without insane inheritance, suffered from asthma from puberty until a few months before an attack of insanity. During this time she lost her asthma, and when she became insane she said the asthma had gone to her brain.

4. A female, aged 64, without insane inheritance, had suffered for twenty years from chronic bronchitis and asthma. She suddenly became excited and free from the chronic ailment. She grew suicidal and violent, but remained free from asthma.

Conolly Norman records the following cases:—

1. A female, aged 45, had had asthma from 25, became insane, and lost the asthma. She remained insane for 2½ years before the asthma returned, since when she continued both asthmatic and insane, but there was no relation between the asthmatic attacks and the mental condition.

2. A male of 50 had been well until the age of 43. He then became suspicious and depressed. These symptoms were all removed by the occurrence of asthma, to which he was ever after liable, but without any return of mental disease.

3. A male of 32 had had almost daily attacks of asthma from childhood. The asthma disappeared for four days, when he became depressed and threatened to kill his mother. For three weeks he continued in the same mental state, when the asthma returned. His mind then became healthy and continued so.

4. A male of 20, always weak-minded and probably asthmatic, became maniacal and lost his asthma for three years. He then became quiet and the asthma returned, but the mental condition remained feeble.

5. A female, aged 40, had been asthmatic all her life. She became suddenly insane, and the asthma left her for six months. It then returned, and the insanity passed away and did not recur.

6. A male, aged 30, had been asthmatic for many years. The asthma became less severe for two years, then delusions set in, and the patient became insane. For 2½ years, while at the worst, the asthma was absent. Then he passed into a chronic demented state, with frequent attacks of asthma.

Kelp[1] records a similar case in a man aged 28, with a history of insanity on both sides. After suffering from asthma for seven years, for which he took large amounts of morphia, he became insane, but free from asthma for seven months. The asthma then returned, and the patient recovered his senses and continued mentally well, but subject to asthma.

One interesting case of the kind has come under my own observation. The whole family is highly neurotic. The mother developed asthma late in life, about the age of 60. One son was a drinker, and two others guilty of the greatest excess both in wine and women, and of these one died of very acute phthisis; one daughter, highly emotional, was asthmatic for more than twenty years, and about the age of 45 passed into a condition of extreme neurasthenic emaciation and prostration, but continued unusually free from asthma for many months afterwards; at the end of which time she had a short attack of acute mania, from which she only very slowly rallied, but all this time remained free from asthma. Some months later she developed phthisis, of which she ultimately died about two years later.

From the time of onset of the symptoms and signs of phthisis the patient was free from asthma and from all nerve symptoms.

Asthma stands in the same relation to hysteria that it does to insanity, *i.e.*, patients may be subject to alternate attacks of hysteria and asthma; but cases of this kind are quite as rare as those of insanity.

Hysterical Asthma.—What is commonly called hysterical asthma is not asthma at all, but a respiratory neurosis of an entirely different kind met with in hysterical patients. The respirations are of very great rapidity, 80 or 90 or even over 100 in the minute, panting, gasping, and noisy, and often more numerous than the pulse-beats. The breathing is deep and the chest expands to the full,

[1] *Ztsch. f. Psych.*, xxiv. 4.

so that the amount of air passing in and out is greatly increased. As the result the cheeks and lips are red and florid, but there is not the slightest cyanosis and no true dyspnœa. The paroxysms are usually of short duration, but may last two or three hours. I have seen one case in which the condition was more or less persistent for many weeks, and in many of the attacks the respirations were considerably in excess of the pulse. These cases resemble most the similar attacks of extreme rapidity of the heart which I have called Paroxysmal Hurry, *i.e.*, one clinical form of Tachycardia.

From the consideration of the affections with which asthma is associated it is easy and natural to turn to the exciting causes.

EXCITING CAUSES.—There is hardly anything which has not in one case or another excited an attack of asthma, and the resemblance of asthma to epilepsy in this respect is so close that the conclusion seems almost irresistible that asthma, like epilepsy, depends upon an unstable condition of the nerve centres, in which the explosion may be excited reflexly by a great variety of causes. Where this centre is to be sought for, whether in the medulla or brain, and whether the instability depends upon an organic, or simply upon a functional, lesion, future investigations alone can determine.

Assuming the existence of such a centre, the paroxysm might of course originate in it spontaneously, but it would be much more likely to be excited by some irritation reaching it from above or below, a view which could be represented in the following diagrammatic form.

1. *From the centre itself.*—In most cases excited by the circulation through it of blood containing some irritating impurity. It seems almost necessary to assume this origin for those cases in which asthma develops in connection with dyspepsia, but not till an hour or two after the taking of food. This is also the probable explanation of asthma occurring in the course of uræmia, though it must not be overlooked that alterations in the tension of the vessels, or other changes in the circulation, may possibly play their part in this affection.

2. *From above.*—To this category would belong (*a*) all those cases which are provoked by emotions, whether of pleasure or pain, by fright or shock, and by immoderate laughter; (*b*) those connected with various cerebral neuroses, *e.g.*, insanity, hysteria, hemicrania, etc., convulsions, and epilepsy; (*c*) those associated with gross disease, *e.g.*, hydrocephalus. Emotion may cure as well as excite the paroxysm. Thus cases are recorded in which the visit to the doctor has stopped the attack, as entering the dentist's house may stop the toothache, and in which a patient, suffering from a severe paroxysm of asthma, was alarmed by her sister suddenly falling down in a faint, when she jumped up and ran for assistance, though a few seconds before the dyspnœa was so severe that movement was impossible.

3. *From below.*—(*a*) Through the special sense nerves. The optic is as rare a source of irritation as the olfactory is common. Bright light often brings on an attack of sneezing, and sometimes an attack of asthma, but the cases more commonly quoted depend rather upon an absence of light than upon an excess, some patients being unable to sleep without a light, and if a paroxysm comes on in the dark, finding relief on lighting a candle.

In connection with the olfactory, we find attacks brought on by odours of nearly every kind—the smell of violets or the rose, of coffee, camphor, or even roast hare; animal emanations from the cat, dog, guinea-pig, and horse; pungent smells or fumes as of pitch or burning sulphur, fog and mist; in some cases by dusts of almost any kind, in others only by the dust of some special substance, of which the most remarkable is ipecacuanha, the fluff of fur or feathers; and here

is the natural place to mention the effect of the pollen of certain grasses to which *hay-asthma* has been referred.

Many of the nasal irritations are, however, connected rather with the fifth nerve than with the olfactory. It is the fifth probably which is at fault where the attacks commence with nasal symptoms, *e.g.*, sneezing, itching of the nose and coryza, and again where organic affections of the nose are present, such as polyp or rhinitis.

(*b*) Through the pneumogastric nerve, either by irritation of its trunk in the neck or within the thorax, or through almost any of its branches, except, perhaps, the superior laryngeal.

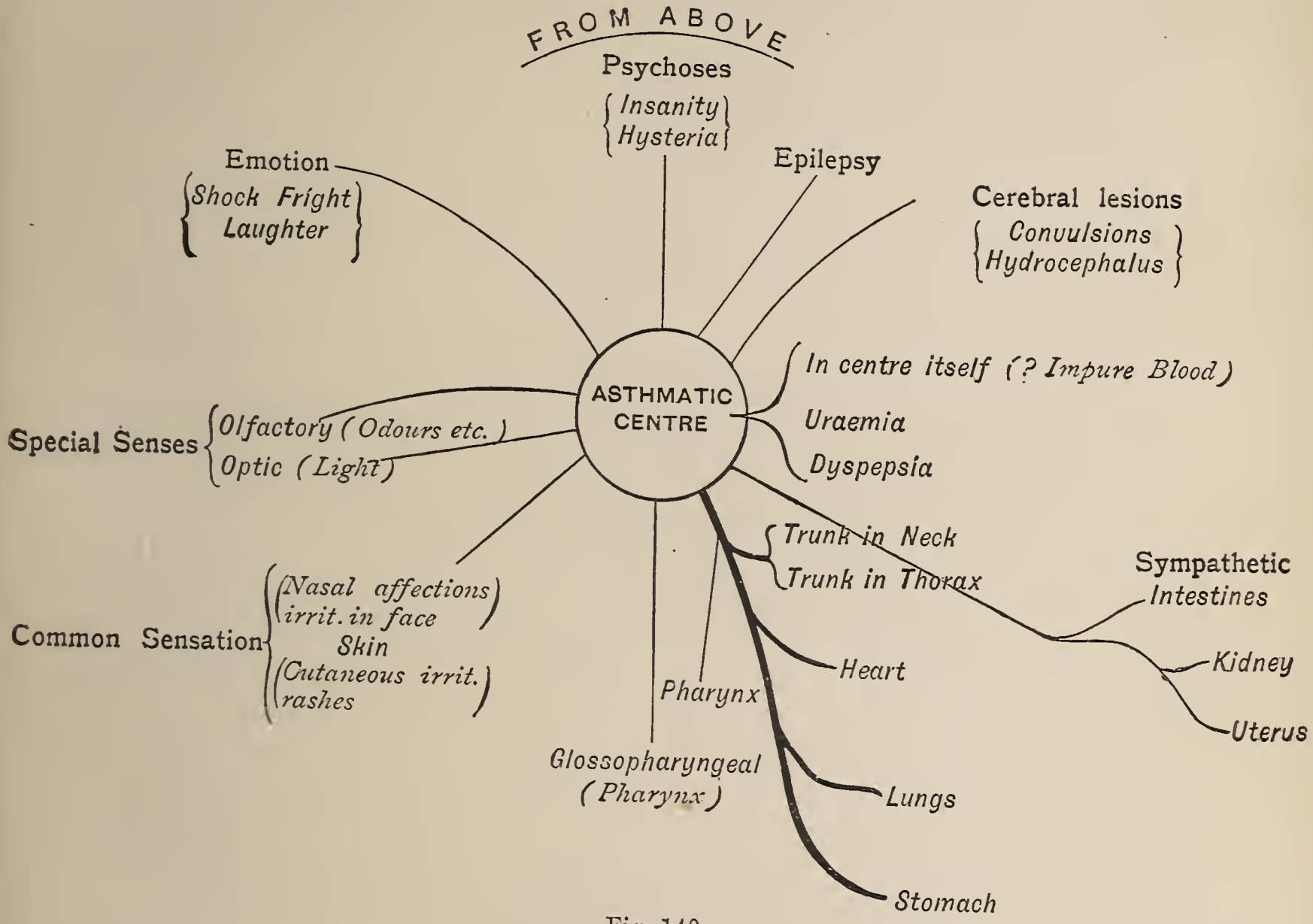


Fig. 140.

Diagram of the exciting causes of asthma.

The pharyngeal, as in the cases quoted of pharyngeal polyp.

The pulmonary, as after the inhalation of dusts, fog, or mist, or after bronchial catarrh.

The gastric, when the attack stands in relation to dyspepsia or to some special idiosyncrasy in regard of certain articles of food, *e.g.*, beer, sweets, etc.

And lastly, the cardiac.

(*c*) Through the sympathetic from the intestines, as with worms and constipation; from the kidney, though uræmia generally, as already stated, belongs to another category; and from the uterus or generative organs.

(*d*) Through the cutaneous nerves, as, for example, after exposure of the face to a cold wind as in riding, cold feet, the application of cold or even of heat (*e.g.*, poultice) to the surface.

Here may be mentioned the remarkable case of a lady in whom the slightest puff of cold air on the back of the neck was sufficient to produce the attack at any time. This case is similar to one referred to by Hyde Salter, in which the attack could be produced by cold water applied to the instep.

A long chapter might be written on the multiplicity and variety of the exciting causes of asthma, but it is evident that if the theory suggested be adopted, the interest shifts from the peripheral exciting causes to the position and nature of the central predisposing condition or lesion if there be one.

THEORY OF ASTHMA.—It has been often necessary, in what has preceded, to assume a theory in order to give a coherent account of the disease. The time has come, now that the facts have been stated, to consider what theory will best explain them.

It may, I think, be taken for granted that asthma is a disease of nervous origin, and that no purely mechanical theory affords an adequate explanation of the phenomena. This is evident from the peculiar origin and nature of the paroxysm, from its sudden access and termination, and from the influence exercised upon it by drugs. In the nervous predisposition which clearly underlies it, and in the great multiplicity of exciting causes, asthma bears a close analogy to epilepsy. If asthma be not a purely functional disease, it is at any rate not associated with any hitherto recognised lesion. It may, therefore, be correctly described as a respiratory neurosis; but it differs from other respiratory neuroses, in being not so much a change in the rate or rhythm of respiration as in its character. As the affection is clearly of a spasmodic character, and is associated with the contraction of muscles, the following questions arise:—

1. What are these muscles which are thrown into a condition of spasm?
2. Is muscular spasm competent to produce all the characteristic symptoms of the disease?
3. Is the spasm excited by direct irritation, or is it a reflex phenomenon?
4. If a reflex, what is its mechanism?

The muscles that may be thrown into spasm are the bronchial muscle, the diaphragm, the ordinary and extraordinary muscles of respiration, and lastly, perhaps some of the muscles of the larynx.

The local phenomena that require explanation are the wheezing, the prolonged expiration, the rigid expansion of the thorax, the low position and defective movements of the diaphragm, the emphysema of the lung, and the secretion from the air-tubes.

A. Spasm of the Bronchial Muscle.—Here, again, several questions arise—(1) Does the bronchial muscle exist, and if so, how is it innervated? (2) Can it contract so as to produce obstruction? (3) If so, can it produce the necessary symptoms?

1. The existence of the bronchial muscle has been denied, but it is now established, and it is innervated from the pneumogastric nerve; but it is interesting to remember that the theory of bronchial spasm antedates by a long time the discovery of the muscle.

In the trachea and large bronchi the muscular coat is well developed, and while in the trachea its contraction might narrow the tube considerably owing to the cartilages being incomplete, in the large bronchi the complete rings would go far to prevent this result. The chief effect would be in the small tubes, where the muscle is known to exist in sufficient amount, and the cartilages are scanty or absent. The nerves supplying the muscle are the vagus and the sympathetic, the former being sensory as well as motor.

2. Can the contraction of the bronchial muscle be demonstrated? Upon this question the results of physiological experiment were for a long time uncertain.

Thus Longet, by stimulation of the vagus, and C. B. Williams, by chemical irritants applied directly to the surface of the bronchi, proved to their own satisfaction that the bronchi could contract. Donders, Wintrich, and Rosenthal,[1] however, repeating the experiments, failed to convince themselves of that fact. Rugenberg attributed the positive results of his experiments to a contraction set up, not in the bronchial tubes, but in the œsophagus. The question may be now considered finally settled by the observations of Bert, Schiff, Gerland, M'Gillivray, and, lastly, by those of Riegel and Edinger.[2]

In all cases the general method of experiment was the same—a tube connected with a manometer was fixed into the trachea, both pleural cavities were then laid open, and the rise dependent upon the elasticity of the lungs registered; the vagus was then stimulated, and the further rise obtained noted. By pushing the tube to the end of the trachea, the contraction of the tracheal muscle was eliminated.

These experiments showed that although the bronchial muscle did contract under the stimulation of the vagus, still that the force exercised by it was small, and not more than about one-fifth of that exercised by the elasticity of the lungs.

3. Is bronchial spasm alone sufficient to explain all the symptoms of asthma? *A priori* it would appear almost certain that a widespread contraction of the small air-tubes, even if it were but slight in each individual bronchus, would produce considerable obstruction to the entrance of air, but that the obstruction would be inspiratory, and not, as in asthma, expiratory.

This difficulty is met by those who, like Biermer, hold the simple theory of bronchial spasm, by the argument that the small air-tubes which are in a state of spasm are subject, like the alveoli, to the pressure of expiration, and being free from cartilages, are in consequence pressed on and still further occluded.

The objection that the emphysema would not be produced by bronchial spasm is fairly met by pointing to the emphysema which accompanies bronchitis in children, where there can be no doubt about the bronchial obstruction.

Bronchial spasm may thus be held to explain, though not altogether satisfactorily, the dyspnœa, the emphysema, and perhaps the wheezing, but it does not account for the secretion, nor entirely for the rigid position of the thorax, nor for the low position and defective movements of the diaphragm. It has been, therefore, assumed that to the bronchial spasm is added some hyperæmia or actual swelling of the bronchial mucous membrane, whether concomitant with the spasm or the result of it. Such hyperæmia has been stated to have been actually observed by Stoerk in the larynx and trachea as well as in the nose.

The secretion in asthma, however, is entirely unlike that which is met with in nervous nasal catarrh, being viscid and scanty, and not profuse and watery. If swelling of the mucous membrane play any important part, it in all probability depends upon a vasomotor disturbance, and is therefore, like the asthmatic paroxysm itself, of nervous origin.

Some doubt may, therefore, be fairly permitted whether bronchial spasm, even with the addition of vasomotor congestion, is adequate to account completely for the phenomena of asthma.

B. Spasm of the Diaphragm.—Because of the divergent results of physiological observation in respect of the bronchial muscle and because of the low position of the diaphragm, which bronchial spasm did not seem competent to explain, Wintrich proposed the theory that asthma was due to spasm of the diaphragm, and this theory Bamberger adopted. It is, of course, a fact that during the paroxysm the diaphragm stands low and moves but little, and that as the paroxysm passes off, the movements increase and the diaphragm returns to its normal position.

[1] For reference, *cf. Hermann's Phys.*, iv. *a*, 243.

[2] *Ztsch. f. klin. Med.*, v. 413.

Those who hold the simple theory of bronchial spasm explain these phenomena as the result of the emphysema, this in turn depending upon the bronchial obstruction. The objections to the theory of spasm of the diaphragm are that it is difficult to conceive of a spasm of the diaphragm which can last as long as the paroxysm of asthma often does; that, even if it occurred, it could not of itself produce the peculiar dyspnœa of asthma; and lastly, that experimental and pathological spasm of the diaphragm gives other symptoms.

In this respect Riegel and Edinger's experiments are of interest. They showed that stimulation of the vagus—of the trunk of the intact nerve, or of the central end if the nerve were divided—produced contraction of the bronchial muscle, and at the same time also a lowering of the edge of the lungs, due to contraction of the diaphragm, which continued as long as the stimulation was maintained. This they found to be a reflex through the phrenic from the vagus, for it was absent when the phrenic was divided. In order to test the relation of the emphysema and the depression of the diaphragm to bronchial hyperæmia and swelling, *i.e.*, to bronchial obstruction, the animals were made to inhale strong ammonia; the same results were observed, but as they were no longer obtained after the phrenics had been divided, it was evident that they were reflex phenomena through the phrenic and the result of irritation of the vagus fibres in the air-tubes. In spite of the rapid dilatation of the lung, which irritation of the vagus produced, no dyspnœa occurred, and they were led to the conclusion that besides spasm of the bronchial muscle and of the diaphragm, determined by irritation of the vagus, some other factors were necessary for the production of asthma, and they looked for these factors in some vasomotor disturbance in the bronchial mucous membrane.

C. Spasm of the other Inspiratory Muscles.—It is quite possible that the fixed and rigid position of the thorax is due to spasm of many of the extraordinary muscles of inspiration, which raises the ribs, and thus prevents expiratory contraction of the thorax. At any rate the raising of the shoulders and the distension of the lower part of the chest are among the earliest phenomena of an attack, and often precede by some little time anything like severe dyspnœa.

D. Besides all this, it is possible that the muscles of the larynx are in a condition of partial spasm, for the stridulous breathing is in all probability, in part at any rate, produced in the larynx, and may be due to the defective widening of the glottis on inspiration, which Steavenson professes to have observed.

It seems impossible, after what has been stated, to arrive at any other conclusion than that the spasm of asthma is a very widespread one, affecting many sets of muscles, the bronchial perhaps first and foremost, but still not alone, and, as well as the bronchial, also the diaphragm and the other inspiratory muscles, including, perhaps, some of the larynx. To this widespread spasm may be added, as an accessory, more or less vasomotor disturbance leading to hyperæmia or actual swelling of the bronchial mucous membrane, which may increase the symptoms, or even, as in the catarrhal form, be predominant.

Such an extensive spasm of so many different muscles can only be of central origin. It must be in the great majority of cases of a reflex nature, and, in support of this theory, all that has been said in relation to the exciting causes of the attack comes in. The irritation which excites the spasm may originate in almost any centripetal nerve, from the pneumogastric in the first place, through almost any of its branches, except, perhaps, the superior laryngeal, through the olfactory or optic, through the fifth or common cutaneous nerves of the skin, and through the sympathetic.

To sum up, then, asthma must be regarded as a reflex neurosis, the symptoms of which are spasm of the bronchial muscle, of the diaphragm and other inspiratory muscles, associated in many cases with more or less of vasomotor disturbance in the bronchi.

This theory necessitates the assumption of a peculiar unstable condition of the respiratory centres, the nature of which is at present unknown, but which constitutes the essential predisposing cause of the disease, and which in all probability is the only heritable part of it.

In all this the resemblance of asthma to epilepsy is very close, and Hughlings Jackson[1] does not shrink from curtly defining asthma to be "a respiratory convulsion," and I presume that he regards it as possibly in many cases a congenital affection, for he proceeds to attribute it to "the discharge of imperfect (? small) respiratory centres."

It is interesting to find that this theory brings us back to the view propounded originally in the *Zoonomia* by Darwin, who stated that asthma convulsions had the same character as all other cramps and epilepsies, and that it could originate, like them, from nearly all distant parts of the body.

VARIETIES OF ASTHMA.—According as asthma is found in connection with other affections or not, it has been described as symptomatic or idiopathic, and of the symptomatic group Hyde Salter, who is responsible for the classification, gives four kinds—the bronchitic, gastric, cardiac, and renal. It may be convenient to make this distinction, in order to draw attention to the affections with which asthma may be associated, but it marks a difference which does not really exist. Among the idiopathic group are several which might with equal propriety be put among the symptomatic. It is misleading to speak of these as different forms of asthma, for in all cases alike the asthma is the same; it is the exciting cause only that varies. It would be simplest to discard the term symptomatic altogether, and to use asthma, idiopathic asthma, or spasmodic asthma as the general term, and to indicate by prefixing the words bronchitic, cardiac, renal, gastric, etc., that the asthma is associated with affections of the parts of the body indicated. This applies equally to hay-asthma, for hay-asthma is the same as any other asthma, the only peculiarity being that it occurs in the course of the affection known as hay-fever or summer-catarrh; while hysterical asthma is a misnomer, and is in most cases not asthma at all, but an entirely different respiratory neurosis dependent on hysteria, and, except in its sudden beginning and ending, in no respect resembling true asthma.

Asthma is a term often incorrectly applied to other forms of dyspnœa, which are not true asthma at all, and in this respect the terms bronchitic, cardiac, renal and gastric have led to much confusion. Asthma is a form of paroxysmal dyspnœa, but every form of paroxysmal dyspnœa is not asthma. This distinction is very important in respect of treatment, for many of the remedies which do good in true asthma do harm in other forms of paroxysmal dyspnœa and aggravate them.

DIAGNOSIS.—Asthma is such a peculiar disease, and so unlike anything else, that in ordinary cases no difficulty can be experienced in making the correct diagnosis. The paroxysmal nature of the dyspnœa, its very capriciousness, the extraordinary prolongation of the expiration, and the defective movements of the chest and diaphragm are pathognomonic.

In *emphysema* the movements of the chest and diaphragm are restricted, and the thorax is widely dilated, but the expiration is not prolonged to the extent it is in asthma, the shortness of breath is permanent, and the exacerbations which occur are due to bronchitis.

From *bronchitis* asthma is distinguished by the severity of the dyspnœa and by the want of relation between the amount of dyspnœa and the physical signs.

[1] *Brit. Med. Jour.*, 1887, i. 159.

In bronchitis the dyspnœa begins and ends gradually, and follows the catarrh, and does not precede it. At the same time both diseases may be present, and the bronchitis may thus mask the asthma. This is especially likely to create difficulty in children, in whom asthma does not suggest itself so naturally to the mind, and in whom bronchitis and its results lead to so much more serious dyspnœa than in adults.

The diagnosis between bronchitis and asthma is of especial importance in respect of treatment, for the inhalation of depressing drugs, which are so useful in asthma, often aggravate and keep up bronchitis.

I have recently had under my care a lady of 40 years of age, in whom the diagnosis had been made of bronchitic asthma. She had been treated in accordance with the diagnosis in the usual way for asthma for months, and had steadily got worse.

An attack of spasmodic asthma in the strict sense of the term she had never had, and her dyspnœa disappeared as the bronchitis subsided.

What the patient was really suffering from was recurrent bronchitis, with a good deal of shortness of breath, worse at some times than others, in relation to the amount of bronchitis.

The stopping of all the depressing remedies and irritating inhalations she had been patiently persisting with, and the treatment of the case as one of bronchitis, rapidly sufficed to relieve the symptoms.

From all forms of *laryngeal obstruction*, such as simple or membranous laryngitis, œdema of the larynx, and stenosis, the diagnosis is made by the character of the dyspnœa, by the violent movements of the larynx and diaphragm, and by the extreme respiratory recession of the softer parts of the thorax. The same applies to the impaction of a foreign body in the larynx or in the trachea, except that in the latter case the movements of the larynx are not violent. If the foreign body be in one of the bronchi, the dyspnœa is less; and the physical signs being unilateral, suffice to give the diagnosis, for unilateral asthma does not occur.

In all these cases alike the difficulty would only arise during the paroxysm, so that the history and the course of the case would be sufficient to make the diagnosis clear.

There remains one other condition with which asthma is not infrequently confused, viz., *plastic bronchitis*; not because the diagnosis is in itself difficult, but because plastic bronchitis is not directly suggested, and is therefore not thought of.

I have seen several instances of this mistake in cases which have been sent to me with the diagnosis of asthma, but which were proved without doubt to be plastic bronchitis by the discovery of the characteristic casts.

In well-marked cases of plastic bronchitis, attention is at once attracted to the peculiar character of the expectoration, and the diagnosis is easily made; but in slighter cases the recurrent attacks of dyspnœa may closely resemble asthma in history, though not in character.

Thus a woman of 40 was under my observation, in whom the diagnosis of asthma had been made. She had suffered from paroxysmal attacks of dyspnœa for eighteen months, which came on regularly every night at 2 a.m., lasted about two hours, and ceased after a fit of violent coughing and the expectoration of viscid sputum. The occurrence of the initial paroxysm so late in life, and the remarkable periodicity of the attack, led me to examine the sputum with care, when the characteristic casts were discovered.

I have seen lately two other similar cases, the one in a man of 36, the other in a man of 55. Both were completely and rapidly cured by iodide of potassium.

PROGNOSIS.—The prognosis of asthma must be considered from three points of view—First, in regard of danger to life during the paroxysm; secondly, as to the effect it has upon the duration of life; and lastly, in respect to the prospects of cure.

1. *Danger to life.*—Alarming as the paroxysms appear, they are hardly ever fatal, and never unless as the result of some complication. Fagge, it is true, records the case of an active and healthy young man who ceased breathing during a paroxysm, and would have died had not skilled assistance been at hand to perform artificial respiration for some time. So far as I know this case is unique.

2. *Duration of life.*—Nor does asthma tend necessarily to shorten life, except so far as it leads to permanent bronchitis and emphysema. Asthma is compatible with long life, and many chronic asthmatic patients live to a good old age and die of some other ailment at the last. Salter records two patients who died at the ages of 69 and 70 respectively, having each been asthmatic for no less than sixty-four years. Still there is always the risk, if asthma continue for long, especially if it frequently recur, that permanent emphysema and chronic bronchitis may result, and the prognosis will then depend upon the amount of these affections that are present.

3. *Prospect of cure.*—This depends upon the age at which the disease developed, upon the frequency and duration of the attacks, upon the length of time the disease has lasted, upon the exciting cause, upon the absence of heredity, and of organic lesions.

If the asthma first appeared in childhood, it may disappear at the time of puberty; if at the time of puberty, it sometimes disappears at the menopause in women, or at a corresponding time in men; if in middle life, it will probably persist throughout life; and if in advanced life, depending, as it often does, upon organic lesions, it will almost certainly continue. Salter puts the facts shortly thus:—If asthma develop before 10, it will probably be cured; if before 20, it may perhaps; if between 20 and 40, probably not; if after 60, certainly not.

The more frequent the attacks, the more severe they are, the longer they last, and the greater the duration of the disease, the less likely is the liability to disappear.

The prospects are better where the attacks are becoming less severe, and where the intervals are growing longer.

The more evident the exciting cause, and the more easily it can be avoided, treated or removed, the better the prognosis.

Heredity has its importance, those cases being best which have no family history of asthma, or of any of the allied affections; but in inherited cases the asthma may disappear, as it may have commenced, at the same age in child and parent.

With organic disease the prospects of cure are small, but a difference holds here according as the organic disease is the cause or the consequence of the asthma; for it not rarely happens that, with the development of some secondary mischief in the lungs, the asthmatic tendency diminishes and may entirely disappear.

To sum up, the prognosis is good, if the patient be young, the chest sound, the attacks short, and the intervals long; if there be no permanent shortness of breath, cough or expectoration; if the attacks are getting milder; and if the exciting causes are clear and avoidable.

TREATMENT.—The objects to be aimed at in treatment are, first to prevent, if possible, the disease from developing; secondly, to control the paroxysm, and thirdly, to check its recurrence.

Prophylaxis.—As far as prophylaxis goes we can do but little, for usually the first indication of a liability to the disease is the occurrence of a paroxysm more or less typical. All that can be done in persons belonging to an asthmatic

family is to protect them as far as possible from the common predisposing causes of the disease, such as bronchitis and hay-fever, and to take more care of them when so attacked. Unfortunately the difficulty of prophylaxis is rendered still greater by the facts (1) that asthma on the one hand often develops in a family in which it is not hereditary, and on the other remains absent where it is markedly hereditary; (2) that it may long lie dormant, and be roused into activity quite unexpectedly by slight causes, such, for instance, as a casual change of residence.

Treatment of the paroxysm.—The paroxysm is, as a rule, too severe, and its treatment too urgent, to allow of much investigation into the exciting causes and associated affections. All that can be done at the time is to give what relief one can, and wait till the attack is past to complete the examination of the case.

In the treatment of the paroxysm we shall be greatly influenced by the theory we adopt of the disease. Accepting the view that asthma is a widespread muscular spasm, emanating from the respiratory centres in the medulla, in response to some peripheral irritation, we have three obvious courses open—(1) to reduce the excitability of the centres; (2) to relax the muscular spasm; and (3) to diminish the peripheral irritation.

The central excitability might be reduced by the use of anæsthetics, such as chloroform or ether, of narcotics like opium and morphia, or of other sedatives, such as chloral and bromide of potassium. Possibly the excessive excitability of the centres may be connected with some defect in their circulation, and thus the action of drugs like caffeine and alcohol, which stimulate the heart, or of pilocarpine and nitrite of amyl, which dilate the vessels, be explained.

The peripheral irritability might be allayed by the use of local sedatives, *e.g.*, cocaine, aconite, and belladonna, where the source of irritation is within reach, as in the nose or pharynx, or of sedative inhalations, where the bronchi are the seat of irritation.

The muscular spasm suggests remedies which relax spasm elsewhere, *e.g.*, chloroform among anæsthetics; or general depressants, such as ipecacuanha, antimony, aconite, and lobelia.

Many of the remedies found most useful, act in all probability in more than one of the ways indicated; for instance, opium is a central as well as a peripheral sedative, and belladonna, while it stimulates the respiratory centre and diminishes the irritability of the peripheral nerves, also relaxes muscular spasm.

Whatever the general indications for the use of this or that drug in asthma may be, our knowledge of their action is to a very great extent empirical, and the utter capriciousness of asthma is in no respect better demonstrated than in the uncertain action of drugs, some drugs being on the whole more likely to succeed than others, but every one failing in turn, so that the treatment of asthma in some cases seems to resolve itself into the trying of a string of remedies one after the other, in the hope that one will be found to succeed at last.

At the same time those physicians are most successful in the management of asthma who study their cases most carefully, and search for the indications which each case offers. General rules are of general application only, and each case of asthma requires to be studied separately, and then success is often achieved where random treatment has failed.

It will be more useful, without attempting too much in the way of classification, to discuss in turn the remedies which have stood the test of experience. They may be roughly arranged into the following groups—sedatives, depressants, antispasmodics, counter-irritants, and stimulants.

The methods of administration are by the mouth, by subcutaneous injection, and by inhalation.

Sedatives.—The sedative group contains stramonium, conium, hyoscyamus, chloroform, ether, chloral, bromide of potassium, cannabis indica, and morphia. Of these, two stand out prominently before the rest, namely, stramonium and morphia.

Stramonium.—Datura stramonium belongs to the botanical family of the Atropaceæ, and all the pharmacopœial drugs of this group, viz., belladonna, stramonium, hyoscyamus, and tobacco, have a powerful therapeutic influence upon the asthmatic paroxysm.

The first three drugs contain the same or closely similar alkaloids, upon the presence of which to a very great extent their activity depends. From stramonium is obtained an impure alkaloid called daturine, but this is found to be an indefinite mixture of atropine and hyoscyamine. Hyoscyamine, further, is isomeric with atropine, though not identical in constitution, and has a similar though less powerful action.

Atropine stimulates the respiratory centre, and at the same time lessens the excitability of the peripheral endings of the vagus in the lungs; it also paralyses the involuntary muscles as well as the nerve-endings in them. Besides this, it has a direct action upon the vagus and vasomotor centres, small doses stimulating and large doses paralysing them. What has been said of atropine applies also to hyoscyamine.

The action of these drugs upon the nerve-endings of the vagus in the lungs explains why their effect is so much greater when burnt and the fumes inhaled, than when introduced into the body in any other way.

Stramonium leaves contain much less of the alkaloid than the seeds, and it is from the latter that the extract and the tincture are made. It is, however, the dried leaves that are most commonly employed, a small quantity being either smoked in a pipe or burnt on a dish and the fumes inhaled. If administered by the mouth the extract is commonly employed, and a quarter of a grain given at once and repeated in an hour's time, but if given early enough, a quarter of a grain may suffice to cut short the attack. Of the tincture, 10 to 30 minims are given at once and repeated in an hour. Internal administration of this drug is more suitable for the intervals than for the paroxysm.

Stramonium was first introduced into this country from India in the year 1802, the Datura tatula being employed, but this was almost at once replaced by the Datura Stramonium, which is now the only pharmacopœial drug. The Datura tatula is said to be the stronger drug, as it contains more of the alkaloid, and it sometimes succeeds where stramonium has failed.

Belladonna and Atropine.—From what has been said it might be supposed that these drugs would have been quite as useful, if not more so, than stramonium, but this is not found to be the case; hence it is not unlikely that stramonium contains some other active remedy not present in belladonna upon which its efficacy depends. Internally belladonna may be given as the extract (gr. $\frac{1}{3}$), or the tincture (ℳ 15 to 20), for a dose repeated at short intervals during the paroxysm; or a subcutaneous injection of atropine (gr. $\frac{1}{100}$) may be given, and repeated in like manner. The most common method of administration is by burning the leaves and inhaling the fumes either alone or in combination, as in the ordinary asthma powders.

Hyoscyamus is weak in action compared with either of the preceding drugs, and is rarely given alone. The alkaloid may be injected *sub cutem* (gr. $\frac{1}{60}$), or administered by the mouth in doses of $\frac{1}{30}$ to $\frac{1}{2}$ grain frequently repeated. This drug, however, rarely succeeds when the others have failed.

Conium has been administered usually in combination with hyoscyamus, but it has even less influence. Recently the hydrobromate has been advocated in doses of one-third of a grain slowly increased up to two grains.

A favourite pill for use at bedtime is the following:—Extract of stramonium, gr. $\frac{1}{3}$; extract of belladonna, gr. $\frac{1}{2}$; and extract of conium, gr. 2 or 3.

Chloroform is of great efficacy during the paroxysm, but its effect is unfortunately transient, so that its inhalation has to be repeated. This can be done without difficulty, for the effect upon the spasm is produced before anæsthesia occurs.

In some cases patients are allowed to administer it to themselves, which they can safely do in the following way:—A tumbler is held in the hand containing some lint or wool moistened with chloroform, from which the patient inhales. As soon as anæsthesia commences, the hand falls and the inhalation stops, to be renewed when the consciousness is restored. Of course an attendant must be present, and the patient must on no account be allowed access to the stock bottles, for accidents may easily occur. One of the great drawbacks is that a craving may be established and the drug used when it is not necessary.

The rubbing of the chest with chloroform liniment is of no more use than a rubbing with any other counter-irritant, and what good it does is more to the bronchitis than the asthma.

Chloroform given by the mouth acts as a stimulant and not as an anæsthetic.

Æther is rarely used as an inhalation in asthma, but is often given internally, when it acts as a stimulant, and in urgent cases it may be injected *sub cutem*.

Chloral.—The relation between chloroform and chloral led to the early introduction of chloral into the treatment of asthma. It has to be given freely, and is best administered in small doses repeated at short intervals, 5 or 10 grains every hour, commencing with a double dose until its effect has been produced. Chloral has a depressing action upon the respiratory centres and also upon the heart, and this makes it unsuitable in cases where the heart is weak.

Cannabis Indica is more useful for the restlessness which sometimes follows an attack than for the paroxysm, upon which it has little or no effect.

Iodide of Ethyl.—There is some doubt as to whether this drug has any anæsthetic action, but containing, as it does, four-fifths of its weight of iodine, it affords a ready means of bringing the body rapidly under the influence of iodine. It is administered by inhalation of 10 to 15 drops at a time, and is found most useful in those cases which are associated with much bronchitis.

Bromide of Potassium is slow in action, but, when given in combination with chloral, is a valuable remedy, especially in a prolonged attack, as it then tends to diminish the severity of the recurrent paroxysms. It should be continued in such cases for some days after the attack is past.

Cocaine.—This drug is chiefly useful as a local anæsthetic, and especially in those cases of asthma which are associated with some affection of the nose, but it has been recommended for use as a subcutaneous injection (gr. $\frac{1}{2}$ of the salicylate of cocaine for the dose). I do not advocate it, for I consider it risky.

Morphia and Opium.—I mention these drugs last because it is wise to put off their use as long as possible lest the opium habit be developed. There is no drug which has so powerful an effect upon the asthmatic paroxysm, and in this respect it ranks even above stramonium, a subcutaneous injection of a quarter of a grain often sufficing to cut short the severest paroxysm. Still it is the drug we have recourse to last of all, for it is often found that increasing doses are necessary, or are thought to be, as time goes on, and thus the morphia habit is engendered. Many patients prefer the suffering of the asthma to the risk of becoming a slave to the opium habit. It sometimes happens that, with self-restraint, the same dose suffices for many years, without any increase, to keep the attacks under control.

I know one case of this kind in which life has been made bearable by the use, when necessary, of a quarter of a grain of morphia, but it was reserved only for the severest paroxysms, the slighter ones being dealt with in other ways.

Morphia has also the disadvantages that its use is sometimes followed by so much depression and debility that the after effects are almost as much dreaded as the relief from dyspnœa at the time is welcomed. In other cases nausea or even vomiting is produced and the appetite entirely destroyed, so that the nutrition suffers profoundly.

This occurred in the case of a lady as the result of one quarter of a grain taken daily *sub cutem*. She became at last extremely emaciated and feeble, so that her life was in real danger. At last the morphia was abandoned and the symptoms disappeared. The difficulty in this case was to convince the patient and doctors that so small a dose of morphia could produce so much harm, and nothing short of the actual demonstration of immediate recovery on abandoning the habit was sufficient to prove it. Since then no persuasion or necessity will reconcile the patient to touch it.

Some of the disadvantages mentioned can be removed by the combination of atropine with the morphia, and this combination is especially useful where, with the asthma, there is much bronchitis, the atropia checking the amount of secretion while the morphia soothes the spasm.

For the paroxysm, morphia *sub cutem* is employed almost exclusively, but a dose of opium or morphia administered by the mouth at night is often sufficient to prevent an attack.

Opium is also a constituent of many of the cigarettes and powders, but in the crude form it is hardly ever smoked, except in combination.

Depressants.—Most of the remedies in this group belong to the class of nauseating emetics, viz., ipecacuanha, tobacco, and lobelia. It is obvious that where the asthma is due to the presence of undigested food in the stomach or to secretion in the air-tube, an emetic would be useful; but if it were the vomiting alone which were efficacious, the simple emetics would be equally active, and this they are not. The secret of their effect lies not in the emesis, but in the depressing nausea produced, for the relief is felt as soon as the nausea is experienced, even when no vomiting occurs.

Ipecacuanha may be administered as the powder or the wine; 20 grains of the powder taken immediately after a draught of half a tumbler of warm water will produce vomiting in about fifteen minutes.

The use of the powder is often followed by prolonged retching, due, it is supposed, to some of the powder adhering to the coats of the stomach and acting as a mechanical irritant. To obviate this the wine is employed, as it is easier to manage, and by graduated doses any degree of nausea and depression may be maintained.

Among the other simple depressants advocated are *Antimony* and *Aconite*, but their use is rather for those complicated cases in which there is catarrh and fever, unless the antimony be added to the ipecacuanha to increase its emetic effect.

Pilocarpine may be also put into this group; at any rate, in the cases in which I have seen it do most good its use has been attended with a good deal of depression.

In one case the injection of one-sixth of a grain *sub cutem*, followed in a quarter of an hour by a second injection of one-twelfth of a grain, produced such alarming cardiac depression that I hesitated to employ it again in that case.

What suggested its use in another case was the spasmodic contraction and high tension of the pulse which accompanied the paroxysm. The first effect observed after the injection was that the pulse lost its hardness and high tension, and the patient immediately cried out, "It is going off." Whether the condition of the pulse was the cause or the effect of the asthmatic paroxysm it is impossible to say, but they disappeared together.

Pilocarpine is a strange drug, and when administered for asthma often does not produce its ordinary action. It relieves the spasm, but does not lead to sweating. In its after effects it probably does good by promoting secretion from the bronchial tubes, and therefore it is best fitted for cases of asthma associated with viscid secretion.

It is best administered by subcutaneous injection in doses of one-twelfth to one-sixth of a grain.

Antispasmodics.—A similar effect to that of pilocarpine upon the vessels follows the use of nitrite of amyl and also of nitroglycerin, but these drugs belong rather to the group of antispasmodics than of depressants.

The *Nitrite of Amyl* is usually given by inhalation, a capsule containing 5 to 10 drops being broken upon a handkerchief and inhaled, or a few whiffs taken direct from the bottle. If given internally the following prescription is useful:— Nitrite of amyl, 3 to 5 minims; glycerin and alcohol, equal parts up to a drachm.

The *Nitroglycerin* is best given in the form of the tabloids of the Pharmacopœia, each containing one-hundredth of a minim.

Other antispasmodics which have been employed are castor, camphor, assafœtida, and ammoniacum, but their action upon the asthma is more than doubtful. If they have any effect at all, it is upon the bronchitis with which the asthma is accompanied.

Counter-irritation.—If this is to have any effect upon the asthmatic paroxysm, it must be of such a nature as to produce a strong shock.

This is the probable explanation of the effect of heat, *e.g.*, the plunging of the hands and arms into very hot water, and the application of a hot strong mustard poultice between the shoulders or in front, or the use of cold, *e.g.*, the application of ice to the chest or spine. These remedies, however, more often fail than succeed.

Stimulants.—Some cases respond best, and sometimes only, to stimulants, and this is the more strange considering that most of the stock remedies have a depressant action. Thus a strong glass of grog at night may avail to keep off an attack better than any other remedy, and may succeed where others have failed.

Coffee; Caffeine.—Strong and hot black coffee is a time-honoured remedy, and its efficacy may be increased by the addition of a teaspoonful or two of brandy to the cup.

Caffeine and citrate of caffeine act more powerfully. I have seen several cases in which 5 grains of caffeine taken when an attack is threatening, once or repeated in an hour's time, have sufficed to keep it under control, and the effect has not been impaired by long use.

Caffeine is an insoluble drug, 5 grains barely dissolving in the ounce of water. Its solubility may be increased by the addition of a little spirit or chloroform, and in salicylate of soda it may be dissolved in sufficient amount to make a hypodermic solution. Caffeine, 20 grains; salicylate of soda, 17½ grains; water, to 1 drachm (♏ iii = 1 grain).

Miscellaneous Remedies.—Grindelia robusta, the Californian remedy for asthma, is sometimes smoked in cigarettes or burnt with other powders. The liquid extract, also, may be given internally in doses of 10 to 30 drops, repeated every half hour or hour in a little sweetened water or milk, to prevent the resin from separating.

Quebracho is said to be most useful in asthma associated with emphysema. It may be given as the tincture in doses of a drachm repeated every hour. The alkaloid aspidospermine is said to have the same effect in doses of $\frac{1}{60}$–$\frac{1}{30}$ of a grain.

Antipyrin has been also found useful. So too has *menthol*, ♏ i to ii of a 20 per cent. solution.

Ergot has sometimes relieved where there has been much congestion of the lungs.

Electricity has been favourably spoken of; but galvanism is now, by common consent, regarded as useless. Recently the interrupted current has been strongly advocated, the poles being placed one on each mastoid process, for about ten minutes.

Inhalation.—The ordinary and most effectual method of administering many of the best remedies for asthma is by burning and inhaling the fumes.

It is in this way that stramonium, belladonna, tobacco, nitre, hyoscyamus digitalis, and even arsenic are usually employed. With the exception of nitre and nitre papers these drugs are rarely used alone, but generally in combination or mixtures in varying proportions, impregnated with nitre to make them burn readily

Nitre.—When nitre papers are burnt in the open air, they give off dense white fumes. These are best inhaled diluted with air rather than pure. The requisite dilution can be obtained by burning them in the room, or if greater concentration is wanted, the curtains of the bed can be drawn, or the papers burnt under an umbrella spread out over the patient.

The fumes when analysed are found to contain much finely divided carbon, to give a strongly alkaline reaction from carbonate of ammonia, and to contain much carbonic acid and water. Cyanogen and cyanogen compounds which were stated to be present by Eulenberg have been since proved to be absent by Kochs.[1] There thus seems to be really nothing to which a potent action can be assigned. It has, however, been suggested that by combustion the nitre is partly converted into nitrite, which exercises an anæsthetic action like the nitrite of amyl.

Of the use of nitre fumes in asthma there is abundant evidence, although no satisfactory explanation of their action is as yet forthcoming. Whatever the cause may be, a considerable amount of drowsiness is produced, which may come on so rapidly that the patient falls asleep before he has time to extinguish the burning paper. Care must be therefore taken that the paper is lighted in a place of safety, where it can burn itself out without danger.

Nitre papers are prepared by soaking strips of thick white blotting paper in strong solutions of nitre in water (100, 60, 40, or 30 grains to the ounce) and drying them.

Some prefer a mixture of nitre and chlorate of potash, the paper being dipped into a boiling saturated solution of the two. It often happens that the burning of a larger or stronger paper succeeds in giving relief where a weaker has failed.

The salt may be also used as a solid pastile made of nitre, or nitre and chlorate of potash, mixed with lycopodium powder.

The papers should be rolled into the form of a cone or folded like a tent and lighted at the apex.

Compound nitre papers.—The papers prepared in the way described may be scented with aromatic substances like benzoin, sumbul, menthol, etc., which are sprinkled on them before they are dry, but it is doubtful whether their efficacy is in any way increased thereby.

Arsenical nitre papers may be prepared by adding liquor arsenicalis to the solution, or dipping them into a solution of 20 grains of arseniate of potash in half an ounce of water and cutting the paper into 20 slips, each of which will therefore contain 1 grain of arsenic.

Powders.—In order to get powders to burn freely, they must be impregnated with varying amounts of nitre or chlorate of potash. Nearly all the powders used are mixtures, *i.e.*, not simple drugs, but combinations of drugs in varying proportions. The leaves or other parts of the drug are first dried and powdered, and then impregnated with nitre (25 per cent. solution) and again allowed to dry. They then ignite freely, and burn like touch paper.

The various well-known asthma cures like Himrod's, Bliss's Green mountain cure, etc., all contain nitre, and most of them stramonium, associated with one or more of the other drugs named. They may be imitated by the following combination :—Dissolve 2 ounces of nitre in 2 ounces of boiling distilled water, add 2 ounces each of lobelia and stramonium leaves and black tea, all in powder ; mix well, dry, and add 4 drops of oil of anise. The quantity used is half a drachm to a drachm when required. (Martindale's Extra Pharmacopœia.)

Smoking.—The fumes are obtained either by smoking a cigarette or cigar, or from a pipe.

[1] *Clbt. f. klin. Med.*, vii. 40, 1886.

If a pipe be used, tobacco is usually the basis employed. With some asthmatics tobacco alone is sufficient, but with habitual smokers tobacco loses its effect.

For them the pipe is nearly filled with tobacco, and upon the top are placed a few pinches of stramonium (15 grains of the leaf or 7 grains of the root) or of belladonna leaves; or of some of the mixed powders, usually without nitre; or the tobacco, whether for cigar, cigarette, or pipe, may be impregnated with a decoction of stramonium, or with similar preparations of other drugs.

Cigarettes.—They are usually made with nitrated papers in which the place of tobacco is taken by stramonium, belladonna, hyoscyamus or other leaves, or the paper may be impregnated with solutions of these drugs.

A common formula in France is the following:—A decoction is made of 5 grammes each of belladonna, stramonium, digitalis, and sage in 1 litre of water and strained; to this are added 75 grammes of nitre and 40 of tincture of benzoin. A quire of blotting paper is gradually placed in it, and allowed to soak for twenty-four hours, after which it is removed, dried, and cut up into rectangular pieces 10 cm. by 7 (4 inches by 3). These are rolled up to make the cigarette, and the edges fastened with gelatin.

Espic's cigarettes consist of belladonna leaves, 6 parts; hyoscyamus and stramonium leaves, 3 parts; phellandrium aquaticum, 1 part; extract of opium, $\frac{1}{7}$ part, dissolved in a little laurel water. Joy's cigarettes are much like Espic's, but they contain lobelia.

Some of the cigarettes contain arsenic, and are so prepared that each is equivalent to a grain of arseniate of potash. Arsenical cigarettes are often of the greatest service. Sometimes, however, arsenic is actually smoked in the solid form in a pipe.

In one extraordinary case, a lady, who had commenced with a quarter of a grain three times daily, had been in the habit for a long time of smoking as much as three grains of arsenious oxide two or three times a day, the smoke being swallowed. She had been able in this way to keep the asthma, which had obstinately resisted many other forms of treatment, under control.

To obtain the full effect from smoking, the fumes should be inhaled, but they are sometimes swallowed, as in the case described. Mere smoking does not produce the same effect. The objection to inhalation, especially when tobacco is used, is that it acts as a strong local irritant and often excites bronchitis.

I know a young lady who smokes habitually for asthma, and though she obtains relief, she is left with a troublesome bronchitis for two or three days, which she escapes if she has not inhaled the fumes.

Other drugs occasionally smoked are Grindelia robusta and Eucalyptus.

Other Inhalations.—*Pyridin.*—The demonstration of pyridin in tobacco smoke suggested its employment in asthma as a direct inhalation, and, according to Germain See, with very great benefit. He recommended a drachm to be evaporated in a small room, and the patient exposed to the vapour for an hour or so three times daily. His experience leads him to prefer pyridin even to morphia.

Carbonic acid was advocated as an inhalation, but considering how rich in carbonic acid the air in the lungs already is in asthma, the *rationale* of its use is not obvious, and it has been abandoned.

Oxygen seems to have more theoretically to recommend it, but Salter tried it freely and found it useless. Richardson combined it with nitrite of amyl and found that it gave great relief, but it must, he says, be administered at a temperature of 75 to 80 F., a condition not easy to comply with.

Sprays are of little use in asthma, for the defective respiration effectually prevents them ever reaching beyond the larynx. A spray of ipecacuanha was advocated by Ringer, especially where the asthma was associated with bronchitis, but its effect, if any, must be due to what is swallowed.

Leyden also advocated the use of a spray of chloride and carbonate of soda with the object of dissolving the crystals in the smaller bronchi, which he regarded as the exciting cause of the spasm, but he failed to show how the substance was ever to reach the place where the crystals existed.

Pneumatic treatment, either by a portable apparatus or of the pneumatic cabinet, has been advocated, but is more applicable to the treatment of the condition between the attacks than during the paroxysm. Whatever use it may have is due to the effect it may produce upon the emphysema, to which the recurrent asthmatic attacks have led.

Locality.—The effect of locality has been already discussed, but it must be always carefully borne in mind in treatment, for change of residence may be the only means of relief, though it does not cure the disease but rather suspends the liability to attack. So extraordinary is the effect of change, that Salter goes so far as to hold that there is probably no case of asthma which could not be cured if only the proper locality could be discovered. Still, change as change is in itself prejudicial, and what is best for the general health is often worst for the asthma, and *vice versâ*, as evidenced by the relief obtained by residence in the dirtiest and dingiest parts of the smokiest towns. Still, there is no end to the caprice of asthma. Places which prevent asthma in one patient provoke it in another, and it may even happen that the individual idiosyncrasy changes in the course of time.

The warm winter resorts of England are often recommended, like Bournemouth, Hastings, etc., and do most good when bronchitis is a factor in the case, but it is often found that what is best for the bronchitis is worst for the asthma, and *vice versâ*.

Woodhall Spa and Kreuznach have their advocates, and the benefit produced is probably due to the iodine in the waters. Mont Dore in the Auvergne has recently acquired a reputation for asthma, though formerly it was thought to be unsuitable. The springs there yield a very weak mineral water and contain also a minute trace of arsenic. The hot baths seem in most cases to do harm, but the sprays (Salles d'aspiration), in association with the bracing air of the mountain, suit some cases well.

Of the Riviera resorts, Hyères seems to be the best, being some distance from the sea. For the same reason Cannes is recommended. Davos and St. Moritz in the Engadine do good sometimes, but Teneriffe is doubtful, while some patients find the hot dry air of Cairo and the Nile, or the mountains of Colorado, Denver and the Springs suit them best.

The conclusion of the matter in respect of foreign resorts is this, that there is no more certainty about the effect of these resorts abroad than at home. All that can be done is to recommend the place which seems most likely to be of benefit and leave the patient to try it.

It is wild and almost unjustifiable speculation to advise long journeys to asthmatic patients, for there is no foretelling the effect upon the asthma when the patient reaches his destination, any more in Colorado than in Cornwall.

When the paroxysm is past the next thing to be considered is how to diminish the frequency and severity of the recurrence. Now is the time to search carefully for any disease or affection with which the asthma is associated and to treat it, and if these conditions do not at the time require treatment, their existence must be borne in mind when the paroxysm recurs, as they often suggest appropriate remedies.

The exciting causes must be sought for, and if they cannot be removed they must be avoided as far as possible. Where catarrh is the usual exciting cause,

not only must the exposure to the causes of cold be avoided, but every means taken of hardening the system and increasing its powers of resistance. Again, where the attacks are preceded by dyspepsia this must be carefully regulated. The greatest possible benefit is often derived from a very strict regimen and dieting, and this is the secret of some of the most popular asthma cures.

Diet.—Asthmatics can rarely live as they please in respect of food. Most soon learn by experience what they can enjoy and what they must avoid. The morning being the best time with asthmatics, it usually matters little what they take for breakfast, and often also for midday dinner. These two should be the staple meals of the day. After midday a light tea in the late afternoon and a little beef-tea, milk, or some easily digestible equivalent, is all that should be taken till the next morning. Nothing is so likely to provoke an attack as a heavy meal indulged in late in the evening, and supper or late dinner is usually an absolutely prohibited meal.

Certain articles of food sometimes bring on an attack of asthma just as surely as they provoke in some patients an attack of urticaria.

In this respect idiosyncrasy must be regarded, but cannot be predicted. The life should be as uniform as the diet, and the greatest regularity both of hours and meals observed. It is often astonishing what an effect a regular and well-ordered life will have upon the frequency and severity of the paroxysms.

General treatment between the attacks.—The importance of treating all affections with which asthma is associated has been frequently referred to. If the attacks are frequent, the health will certainly suffer somewhat, and most asthmatics are the better for a tonic of some kind, and one of the best combinations is iron and arsenic. There are two drugs which exercise great influence upon the affection, and diminish the liability to recurrence—viz., *arsenic* and *iodide of potassium*. They must both be given in full doses and over a prolonged period of time.

Bromide of potassium is greatly inferior to the iodide, but is sometimes useful.

Colchicum in gouty cases may prove beneficial.

Quinine suggests itself in some of the periodic cases, but it is a disappointing remedy.

Sulphur has been recommended, but its usefulness is rather against the bronchitis than the asthma.

Besides all these, a string of nervine remedies has been employed, but all of them with uncertain results, *e.g.*, *oxide of zinc*, *nitrate of silver*, *valerian* and the *valerianates*, etc., etc.

As most asthmatics are in fair health between the attacks, the general treatment is summed up in giving tonics where they are indicated, and in trying a course of arsenic or iodide of potassium.

History.—Asthma was a term used by early writers simply to mean difficulty in breathing. This Celsus divided into three forms—dyspnœa, where the difficulty was slight; asthma, where it was severe; and orthopnœa, where it was extreme. Aretaeus was the first to restrict the use of the term asthma to a peculiar form of respiratory difficulty, of which orthopnœa was a variety, using for all other forms the term dyspnœa. Galen, however, discarded asthma from his terminology, and spoke only of dyspnœa as the general term and of its varieties, orthopnœa and apnœa. It was not till the end of the seventeenth century that Willis established asthma as an independent disease, though this had been previously indicated by van Helmont, in contrast to earlier writers, by whom asthma was regarded as a symptom only. "Viscera omnia sana praesertim pulmones," was his commentary, and he named the affection asthma convulsivum et spasmodico-flatulentum or asthma spasmodicum.

Floyer, who himself suffered from asthma for thirty years, in his book on the subject, also called it asthma periodicum flatulentum, and he initiated the theory that it was due to a convulsive contraction of the bronchial tubes and bladders of the lung. This theory Cullen adopted in his writings about fifty years later.

Darwin, in his *Zoonomia*, distinguishes between humoral or hydropic asthma, which he regards as a temporary anasarca of the lung, and convulsive asthma, the latter having the same characters as all other cramps and epilepsies, and originating, like them, from nearly all distant parts of the body. He mentions a case in which asthma disappeared with the development of gout, and another in which the attack followed the retrocession of an eruption on the face.

By the beginning of the present century, the belief in the existence of a nervous asthma had gone out of fashion, and asthma had become again merely a form of dyspnœa and a symptom of other affections, especially of bronchitis. The introduction of auscultation showed, however, that in most cases the bronchitis followed, and did not precede, the asthma, and Laennec, though not entirely denying its existence, explained most cases by his catarrhe sec, while his contemporaries, Louis, Beau, and others, absolutely denied the existence of nervous asthma, and, later, Rokitansky referred the symptoms to emphysema.

In 1835 the nervous theory of asthma was revived by Ramadge, who attributed it to spasm of the trachea and bronchial tubes, and compared it with colic of the intestines. Romberg, in 1841, relying upon the discovery of muscular fibres in the bronchi by Reisseisen, adopted the same view, but it was not until Bergson wrote his prize essay in 1850 that idiopathic asthma came at last to be generally recognised as an independent affection.

From this time forward, the existence of spasmodic asthma has been acknowledged, and discussion has turned upon the special mechanism of the spasm. Most authors then referred the spasm to contraction of the bronchial muscle, but the endeavour to demonstrate this experimentally produced the most divergent results, Longet, C. J. B. Williams, etc., being on this point in direct opposition to Donders, Wintrich, Budd, etc.

Wintrich, in 1854, having failed to convince himself of the contractility of the bronchi, sought for the cause of asthma in a tonic contraction of the diaphragm, either alone or in combination with other respiratory muscles. This view Bamberger adopted, and extended by clinical observation. About the same time, and later, Salter, writing in this country, adopted without compromise the view that asthma was due to bronchial spasm only, and in 1870 this theory received the strongest support from Biermer, who based his opinions upon the physiological experiments of Paul Bert. With each fresh demonstration of the contractility of the bronchi by Bert, Gerlach, Schiff, and MacGillivray, the bronchial spasm theory received additional support, but still, admitting the power of the bronchial muscle to contract, the difficulty of explaining satisfactorily all the phenomena of asthma by bronchial spasm remained; and in 1872 Weber was led to suggest as the essential factor in asthma a vascular congestion and swelling of nervous or vasomotor origin, and this view Riegel, while still holding to the theory of bronchial spasm in the main, adopted as part explanation. In 1887, Germain See, whose views had for a long time been tending in that direction, definitely pronounced asthma to be a neurosis of the respiratory centres in the medulla, which resulted in a spasm of the respiratory muscles, and especially of the diaphragm. This view the experiments of Riegel and Edinger upon the pneumogastric nerve confirmed, and recently Hughlings Jackson has not hesitated to describe asthma as a respiratory convulsion, the origin of which is to be sought in the medulla, and this is indeed the view to which an impartial review of the facts seems necessarily to lead.

Among the theories of asthma which have been proposed, and either disproved or not generally accepted, may be mentioned—

1. Leyden's; which referred the bronchial spasm to the irritation set up by the presence in the bronchi of peculiar crystals. These are very frequently present, if not invariably as some hold, but as they have also been found in other diseases without asthma, they are rather the effect than the cause.

2. Curschmann's; according to which the symptoms are due to exudation into the tubes, "Bronchiolitis exudativa." This is obviously insufficient, for in the first place the exudation is not constant in asthma, and when it occurs to a much greater extent, as in plastic bronchitis, it does not lead to the symptoms of asthma. Still it is quite true, that in some cases of plastic bronchitis in the small tubes asthmatic symptoms may arise, and I have recorded such cases myself.

3. Schmidtborn's; in which the cause is sought in a spasmodic contraction of the pulmonary arteries. But no evidence in favour of this view is brought forward, nor can any be at present. It remains a mere speculation, and seems to me to explain nothing.

4. Sir Andrew Clark's; that the cause is to be found in transitory local œdema of the bronchial mucous membrane, similar to that observed in the skin in urticaria. A still better analogy would be with those curious cases which are sometimes called angeio-neurotic œdema.

53. WHOOPING-COUGH—PERTUSSIS.

Whooping-cough is an infectious disease, of which the prominent symptoms are a peculiar catarrh of the upper air passages and a characteristic cough. It is usually epidemic, and is dangerous, chiefly from its complications.

History.—The affection cannot be clearly traced back beyond the sixteenth century, when it was first described by de Baillou (1578) and Schenck (1600). Prior to these times it was confused with bronchitis and with influenza. Willis (1682) describes it as "Tussis puerorum convulsiva seu suffocativa et nostro idiomate 'chin-cough' vulgo dicta," so that, even at this time, it was well-known enough to bear a popular name. In the eighteenth century writings on the subject became more numerous, from which it might be argued that the affection had become more frequent; but it is more likely that the difference of opinion as to its nature had the usual result of drawing general attention to it. Upon the infectiousness of the disease, however, all authors have been agreed from the earliest time, however divergent may have been their theories as to its nature.

In England, the earliest names by which it was known were chin-cough or kink-cough; but whooping-cough has been for a long time the popular as well as the technical term. Coqueluche in France and Keuchhusten in Germany are the names in common use among the people, but Tussis convulsiva, the name given it by Willis, and Pertussis, that given it by Sydenham, are terms by which it is recognised in all countries.

SYMPTOMS.—A well-marked attack of whooping-cough is commonly divided for the purposes of description into three stages—1st, That of catarrh; 2nd, that of the characteristic cough; and 3rd, that of convalescence.

It is obvious that these three stages are not sharply defined, but merge one into the other. In mild cases one or other may even be absent; for example, the affection may not go beyond a catarrh, so that the characteristic cough may fail, or, on the other hand, the cough may be the only symptom and the catarrh be almost too slight to recognise.

1. *The first or catarrhal stage.*—Under ordinary circumstances the first symptoms are those of catarrh of no great severity, which often come on suddenly without apparent cause. In more strongly marked cases the symptoms are those of a feverish cold; the eyes are suffused, the cheeks flushed, the nose stopped up, the voice somewhat husky, and the cough frequent. In the course of a day or two the nose begins to run and the catarrh to spread to the large air-tubes. Though the voice may be husky and hoarse, still there is not, as a rule, any marked laryngitis. In the same way the nasal catarrh is as a rule not severe, but sometimes there may be a copious running from the eyes and nose, with long paroxysms of sneezing.

The cough in these cases of early catarrh, though not characteristic, is very troublesome. It is frequent, short, irritable and persistent, like that of measles, may recur 40 or 50 times in the minute, and being usually worse at night, interfere greatly with rest.

The temperature may be raised for two or three days, but rarely passes above 100 or 101. The pulse is accelerated and so is the respiration. Though in a severe case the child may complain of feeling chilly, anything like a rigor is rarely seen unless there be some inflammatory complication. The appetite is lost, the tongue coated, the bowels usually confined, but sometimes there may be looseness, the head is heavy and aching, and the child is ailing and irritable. In all these symptoms there is nothing characteristic, but when an epidemic of whooping-cough prevails, all catarrhs should be regarded with suspicion, and carefully treated.

Speaking generally, the catarrh in this stage is slight, often so slight as to be overlooked, but when considerable and in a young child, it is of serious prog-

nosis, and indicates a severe and probably dangerous attack. When this stage is sufficiently well marked to measure, it lasts usually a few days, a week or ten days on the average, but its duration varies greatly, even from two to thirty-five days.

2. *The second or convulsive stage.* — The commencement of this stage is marked by the occurrence of the characteristic cough. This may make its appearance abruptly, but more often the cough of the preceding catarrh undergoes a gradual change, becoming less frequent and more paroxysmal, and often ending, even when not especially violent, with vomiting. Such a gradual change is only observed where the early catarrhal stage is well marked, and in some mild cases the first indication of the affection may be the whoop.

The true paroxysm consists in a series of short coughs, or, as they have been well called, expiratory explosions. These follow one another in rapid succession, uninterrupted by any corresponding inspiration, until the chest becomes, as it would seem, almost empty of air, and the child grows blue in the face, and seems on the point of suffocation; then all at once a long-drawn inspiration is taken, which, as the air passes through the still contracted glottis, gives rise to the loud whoop or crow from which the name is derived. During the coughing mucus runs from the nose, tears from the eyes, saliva from the mouth, and the paroxysm finally ends with the discharge of a certain amount of viscid mucus from the mouth, and in many cases with the ejection of the contents of the stomach.

Usually in each paroxysm there is not more than one whoop, but there may be several, and then there is usually after each a short pause before the cough begins afresh.

In a severe paroxysm the distress towards the end of the fit is extreme; the face is blue, the eyes red and starting from the head, the mouth gaping, with the tongue thrust forward between the teeth, mucus dropping from the nose and saliva from the mouth, the veins of the neck and face greatly distended, while bleeding may take place from the nose and even ears, or into the skin and beneath the conjunctiva, urine and fæces be passed unintentionally, and the attack end with vomiting.

The pulse during the paroxysm is accelerated and often irregular or intermittent. In a bad fit it may be so rapid as to be almost uncountable. The temperature in this stage is not raised, unless there be some complication; the extremities are cool, and the body and face often covered with cold sweat. The actual paroxysm is often preceded by the rattling of mucus in the trachea, which may be heard at a distance from the patient, and for some minutes before the cough commences. The child seems conscious of the impending attack; if an infant, it becomes restless, unhappy, and fretful and refuses food; while older children often complain of the rattling and tickling sensation in the throat, or of a feeling of nausea; they stop in their play, run to the nurse or mother, and cling to her for support, or in anticipation of what is to come, rush to the basin. When the paroxysm is over the relief is immediate; the infant will fall asleep again or take the food it had before refused, the older child will return to its play, unless exhausted by a very severe attack, when it may complain of headache, and prefer to lie still for a time or even to sleep.

As the chest becomes emptied of air, the percussion note, it is stated, becomes less resonant. Theoretically this might be so, but it would, under any circumstances, be difficult to demonstrate, and in many cases the percussion is tympanitic, owing to the emphysema which has been produced by the coughing.

On auscultation, all that is heard during the paroxysm is the short expiratory jerks of the cough; during the whoop no distinct respiratory murmur is audible,

chiefly because it is masked by the loud laryngeal sound, but also perhaps because, as Laennec suggested, the bronchi are in a similar condition of spasm to the larynx.

After the paroxysm the lungs are more or less emphysematous, and in infants, just as with bronchitis and for the same reason, the upper parts of the chest in front are prominent and distended, showing the predominance of emphysema there.

The Whoop.—The whoop is due to inspiration occurring while the glottis is still contracted.

The spasm usually stands in direct relation to the severity of the cough, but not invariably, for the cough may be severe without much spasm, and therefore with but little whooping; and, on the other hand, the spasm may continue after the cough is past, and in infants even lead to acute suffocation.

In infants the whoop is not conspicuous, and may not infrequently be absent. This fact explains the different views as to the frequency of whooping-cough in infants, the affection often appearing in them only as a severe catarrh, with a troublesome but not characteristic cough. In older children the whoop rarely fails. The earlier the whoop develops, the more severe the case is likely to prove. The whooping generally continues at its maximum for two or three weeks, and then slowly subsides, but it is liable to recur on very slight cause, even it may be for several months afterwards.

With the occurrence of inflammatory complications, for example bronchitis, and above all pneumonia, the paroxysmal cough and whoop tend to disappear for the time, and to return when the inflammation has subsided. This is especially likely to happen in very young children. The difficulty of diagnosis may then be very great, and it is wise with broncho-pneumonia in little children always to bear in mind, as the possible determining cause, a prior attack of whooping-cough. It is, at all events, not rare in cases of broncho-pneumonia, in which a history of whooping-cough has not been obtained, for the whoop to appear during convalescence, and thus establish the true nature of the disease. On the other hand, it must be borne in mind that a little whoop or crow, as it is called, in infants is by no means uncommon, as the result of flatulence, or an incomplete attack of laryngismus stridulus. The actual paroxysm varies in duration from a few seconds to a few minutes, there being as a rule in each only one or perhaps two whoops. The number of paroxysms varies greatly from one or two during the whole attack to a great number each day. At the height of the disease, it is not uncommon to get paroxysms every hour or even every half hour. A case is recorded (Macall) in which there were 140 in a single day, and that in a child eight months old.

The paroxysms are generally most frequent and worst at night.

The attacks are easily excited at any time by various causes. Chief among these is the taking of food, especially the more substantial meals of the day, the fit being followed by the ejection of the meal just as it had been taken, until the patients dread the very sight of food. The debility and failure of nutrition thus produced may constitute the greatest difficulties in the case.

The vomiting may be sometimes successfully checked and the food retained, by giving it only immediately after a paroxysm. It does not, however, follow that when vomiting takes place after food the whole meal is necessarily brought up. On the contrary, a large part of it is often retained, so that in spite of frequent vomiting the nutrition may be fairly preserved.

Other exciting causes of a paroxysm are movements of the larynx, as in speaking, shouting, crying; pressure over the larynx; violent efforts leading to increased respiratory action, as running, straining, etc.; above all, emotional disturbances such as a fit of temper, crying, or laughing. Lastly, anything which under ordinary circumstances would excite a cough will, in whooping-cough, produce a paroxysm, *e.g.*, changes of temperature, dusty places, crowded rooms, and even, it is said, air laden with carbonic acid or other impurities.

The duration of the paroxysmal stage is, on the average, about four weeks.

3. *The third or convalescent stage* is characterised by the gradual subsidence of all the characteristic symptoms, the case often ending in the way it began as a catarrh. The duration of this stage is usually from three to four weeks, but much will depend in any given case upon its freedom from complications.

The general health in nearly all cases suffers considerably, and there is great loss of flesh, so that the children may be reduced to mere skin and bone. This is due partly to the exhaustion of coughing and the want of continuous sleep, partly to the loss of appetite and the difficulty of retaining food upon the stomach. In little children the bowels often become loose and offensive, and a condition of chronic mucous diarrhœa sets in which is very difficult to control, and not rarely ends in fatal marasmus.

It is not unlikely that the loss of appetite and gastro-intestinal disturbance may be in part produced by the mucus which has been swallowed and undergone decomposition. For it is little children who are especially liable to this complication, and they do not voluntarily expectorate. The clearing out of this mucus from the stomach by vomiting may explain the value attached by some writers to the administration of an occasional emetic, and supports a common popular belief that children who vomit in whooping-cough do well.

Even when all symptoms have subsided and the appetite has returned, the nutrition may not improve. For this state of things there is nothing but change of air to the hills or to the seaside, which often at once results in rapid convalescence.

DURATION.—The average duration of whooping-cough is about two months, but individual cases vary within wide limits, and what is true of the affection as a whole is also true of the different stages.

The total duration is thus given by different authors. Gerhardt, 2 to 10 weeks; Steiner, 3 to 8 weeks; Biermer, 4 to 5 weeks; Ch. West, 10 weeks; Barthez and Rilliet, 7 to 10 weeks.

The total duration is thus divided among the different stages:

	1st Stage.	2nd Stage.	3rd Stage.		Total duration.
Ch. West, . .	2 weeks (2–35 days)	4 weeks	4 weeks	=	10 weeks.
Barthez and Rilliet, .	8–15 days	30–40 days	10–15 days	=	7 to 10 weeks.

COMPLICATIONS.—The chief complications may be referred to the catarrh, to the violent coughing, and to the nervous disturbance.

1. **Complications associated with the catarrh.**—Most of the grave complications arise, as would be expected, in connection with the respiratory organs.

Catarrh of the upper air passages, i.e., of the trachea and main bronchi, is a part of the disease, and it is from this source that the viscid mucus expectorated at the end of the paroxysm is derived. This catarrh may spread upwards and involve the larynx and parts about the glottis, but laryngitis is not as a rule a prominent symptom. In most cases the voice is not even hoarse or husky, but in some the symptoms of laryngitis may be severe enough to raise even the question of croup. In a few rare instances membranous laryngitis has been

actually found, but this is probably nothing more than the accidental association of two entirely independent diseases. On the other hand the catarrh is especially liable to descend to the smaller air-tubes, and the *bronchitis* thus produced constitutes one of the commonest and at the same time one of the gravest of the complications. In little children bronchitis is always serious, and especially so when complicating whooping-cough, because of the increased risks of *collapse* and *broncho-pneumonia.* Fortunately when broncho-pneumonia is severe the cough usually becomes less paroxysmal, and the whoop may disappear, so that the lungs and circulation are relieved, at any rate for the time, of that extra strain upon them.

Pulmonary *emphysema* is very common, and stands in most cases in direct relation to the bronchitis, but the tendency to it is greatly increased by violent paroxysms of coughing.

Collapse at the bases, when there is much bronchitis, may easily be overlooked, for it is by no means rare without any rise of temperature and with no other symptoms than an increase in the dyspnœa.

All these respiratory complications not only increase the danger at the time, but, being very obstinate and liable to recur, protract the illness and prolong convalescence.

The *bronchial glands* are in nearly all fatal cases found enlarged. This is not a primary lesion, as it was formerly regarded, but the necessary consequence of the bronchitis and broncho-pneumonia, from which few fatal cases have been free.

Pleurisy is rare except when occurring over a superficial patch of broncho-pneumonia.

Pericarditis as well as endocarditis have been described, but they are rare, and probably stand in merely accidental association with whooping-cough.

2. Complications associated with the violent cough.—The prolonged attacks of coughing greatly obstruct the circulation of the blood through the lungs, and thus throw a great strain upon the right side of the heart. It is to this that the irregularity and intermittency of its action is to be referred, and some of the cases of sudden death during a paroxysm may well be due to cardiac syncope from over-distension. The results of congestion are more evident in the veins of the head and neck, the large trunks of which become greatly distended, while the small venous capillaries may rupture and give rise to hæmorrhage.

These *mechanical hæmorrhages* are almost exclusively confined to the head and neck, owing no doubt to the ease and completeness with which the main trunks from above can be compressed as they pass through the narrow upper opening into the thorax. That there is nothing in this peculiar to whooping-cough is evident from the fact that similar hæmorrhages are met with in epilepsy where the fits have been severe and frequent. The hæmorrhages have these peculiarities in common, that they are rarely considerable, and that they occur only during the paroxysm and cease as soon as it is past.

Epistaxis is the commonest form. It is rarely profuse, but it may, when frequently recurring, greatly increase the debility and anæmia.

Subconjunctival ecchymosis is also frequent. It is generally bilateral and more or less symmetrical. Trousseau has recorded a unique case in which the tears were blood-stained.

Bleeding from the ears may also occur. This probably never happens without rupture of the membrana tympani, and this rupture is generally bilateral. Roger,[1] in a case of old perforation, saw the blood gush freely out of the hole in the membrane at each paroxysm. In most cases, when the cough is well, the

[1] *Bull. Acad. Med.*, 1879.

membrane heals and the deafness produced at the time by the perforation is completely recovered from.

Ecchymoses into the skin are common enough about the face and neck, but only rare on the trunk and extremities. They are small, punctate, and rarely amount to more than petechiæ. Trousseau saw a nævus over the orbit bleed freely during the paroxysms.

The saliva and mucus of the mouth are not infrequently blood-stained, the blood coming from the gums, from a crack in the lip or tongue, or from an ulcer of the frænum.

From the main air-tubes small amounts of bleeding (*streaky hæmoptysis*) are common enough, but anything approaching to profuse hæmoptysis is very rare, and attributable to some complication.

Hæmorrhage into vesicles of the lung is not, as far as I know, recorded.

Temporary defects of vision, even amounting to amaurosis, are occasionally met with. They are in all probability due to the venous congestion, for they quickly pass off, and I do not know that any instance of retinal or subretinal hæmorrhage actually observed has been recorded.

Intracranial congestion may explain the stupor of some cases and the convulsions of others, and it may also lead to œdema of the brain, or effusion into the ventricles, but *actual hæmorrhage* into the brain is rare.

Another important complication resulting from the violent coughing is *rupture of the lung*. This usually takes place beneath the pleura, and not infrequently at more spots than one, so that bullæ of air are found in many places. From this the air tracks either along the bronchial tubes, or, as is more common, round the root of the lung to the mediastinum, whence it may spread to the subcutaneous tissue of the neck and so become widely diffused over the whole body. Sometimes the pleura gives way and pneumothorax is produced, which is generally fatal, but, short of pneumothorax, many of the cases of rupture of the lung, even when they have led to widespread general emphysema, recover.

Among the mechanical results of coughing may be placed the *Frenal Ulcer*, to which a specific value was formerly assigned. Attention was first drawn to it in this country by Morton,[1] but it had been previously described abroad by Bouchard and others. In little children the distress causes them to thrust the tongue forward at the height of the paroxysm. It is thus rubbed over the teeth, which cut the surface, and thus produce the ulcer.

The ulcer is a shallow abraded surface without definite characters, sometimes quite superficial, sometimes deep, and not infrequently bleeding. It is usually met with in children who have cut only one or two of their lower incisors. It is rare in older children who have cut all their teeth, or even all their front teeth, and it does not occur in infants who have no teeth at all. It is not found before the spasmodic stage is reached, and it may develop then in three or four days, but it is often absent throughout. Its usual seat is the under surface of the tongue, on both sides of the frænum if both lower incisors are cut, or on one side if but one be cut. Sometimes an ulcer of similar origin is met with on the upper surface of the tongue.

Two other complications to which the violent coughing leads, neither of which is rare, are *hernia* and *prolapse of the bowel*, but they call for no special comment.

3. Complications connected with the nervous system.—The most important of these is *convulsions*. The liability to convulsions stands in most cases in direct relation to the severity of the paroxysms, *i.e.*, they are most likely to

[1] *Harveian Soc. Trans.*, 1876.

occur where the paroxysms are severe; but this is not a constant rule, for convulsions may be absent where the fits are severe, and present where they are comparatively slight. Something depends on the age, the liability to fits being greater the younger the child is; and something also upon the constitutional predisposition, for some children have fits on very slight provocation, so that in this respect, convulsions being a serious and often fatal complication, constitutional tendency becomes an important factor in prognosis.

Convulsions form an always grave complication, but their relative gravity in any particular case depends greatly upon their cause. They may be simply an exaggeration of the general nervous disturbance which forms a part of the disease; they may mark the onset of some acute inflammatory complication; they may be the result of gastro-intestinal disorder, or of some cerebral lesion; or lastly, be a suffocation symptom, as in any other form of asphyxia.

Carpopedal contractions and *twitchings of the face or limbs* are also common. They are of the same nature as a fit, and may develop, though not necessarily, into convulsions.

In bad cases the child may become heavy, drowsy, or almost comatose. This, when not the result of an intracranial lesion, is due to carbonic acid poisoning, consequent on some grave pulmonary complication.

Acute meningitis and *hydrocephalus* are described as occasional complications, but it is doubtful if they stand in any close or direct relation to whooping-cough.

Hemiplegia is not altogether rare. I have at any rate seen several instances of it in quite young children.

Thus a child of 3 years of age during a violent paroxysm became convulsed and unconscious. On recovery it was found to be hemiplegic on the right side and aphasic. Motion was ultimately recovered, but athetosis developed on the affected side. Speech was also completely restored in the course of several months.

A similar case is recorded by Beevor.[1]

SEQUELÆ.—Whooping-cough has no sequelæ save those of the complications to which it leads. Thus *chronic bronchitis* and *emphysema*, and even *asthma*, may date back to whooping-cough in childhood. The irritability of the larynx to which the whoop is due may persist, and the child be subject for some time to attacks of *laryngismus stridulus*; but, as after whooping-cough rickets not infrequently develops, or if it has been present before becomes aggravated, the possibility of the laryngismus being due to this cause must not be overlooked. The only sequelæ, if they may be called so, which whooping-cough leaves behind are the *anæmia*, *debility* and *general delicacy*, which may continue for months and be only cured by change of air.

General dropsy has been described as following whooping-cough, but it is not peculiar in any way, and is nothing more than the anæmic anasarca which may develop in a weakly child, cachectic from any cause. It is a rare affection under any circumstances, and especially as a sequela of whooping-cough.

The most serious sequela, though fortunately not a very common one, in weakly children, especially if they are rickety or come of a tubercular stock, is the development of *tuberculosis* in the glands, bronchi, or lungs which have been the seat of lesion during the height of the disease. Whooping-cough may thus lead to phthisis, or even to general tuberculosis.

Post-mortem, tubercular lesions are not rare, but the relative frequency of such lesions among fatal cases must not be taken as the measure of their frequency in cases which do not die, for the children may have been tubercular before, and, if so, would be most likely to die when attacked by whooping-cough.

[1] *Clin. Soc. Tr.*, xx. 96.

PATHOLOGY.—The essential pathology of whooping-cough is unknown. In fatal cases certain morbid changes are frequently found, but they are such as result from the complications, unless the catarrh of the large air passages be regarded as part of the disease. With this exception the lesions are the ordinary ones of bronchitis, viz., collapse of greater or less extent, emphysema and broncho-pneumonia. Occasionally, as the result of coughing, subpleural, interstitial and mediastinal emphysema may be found, or even pneumothorax. Enlarged bronchial glands are the rule, and if not of date antecedent to the attack of whooping-cough, are due to the bronchitis and broncho-pneumonia to which it has led. The glands are sometimes caseous, and the lung may be the seat of tubercular infiltration.

The lesions of rickets are common, and to these may be referred the large liver and spleen occasionally described. This is but another way of saying that rickety children who are attacked with whooping-cough often die.

Ecchymoses are frequent in the skin and conjunctiva, as well as internally in the pleura, in the pericardium, in the bronchi, and even in the lung tissue.

In the brain the veins are congested, and fluid is sometimes found in the ventricles; meningitis has been described, and in other cases actual hæmorrhages into the cerebral tissues have been found.

All these lesions are obviously accidental, and none of them constitutes in any way the essential pathology of whooping-cough, which remains yet to be discovered.

Theories.—The facts which require to be explained by any satisfactory theory of whooping-cough are these—(1) Its contagiousness and epidemic character, and the immunity given by one attack. In these respects whooping-cough resembles the specific fevers. (2) The peculiar character of the respiration, viz., the spasmodic cough and the whoop, which suggest nerve irritation; and, lastly, the local catarrh. The theories, therefore, are chiefly three—(1) That whooping-cough is a zymotic disease; (2) that it is a neurosis; and (3) that it is a specific catarrh. According to the theory adopted, whooping-cough appears in systematic treatises on medicine in different places, either among the general infectious diseases, or among the local diseases of the nervous or respiratory systems respectively.

1. In favour of the zymotic theory are the facts that it is undoubtedly contagious, and has a period of incubation, that it prevails in epidemics, and that one attack almost invariably provides immunity for the rest of life. The chief arguments urged *per contra* are the peculiar nature of the attack, the comparative slightness of the fever and constitutional disturbance in uncomplicated cases, and the long duration of the affection.

2. In favour of a neurosis is urged the peculiar character of the paroxysm, but against it stand all the arguments in favour of the zymotic theory, as well as the fact that whooping-cough differs from other neuroses in not being, like them, liable to recur.

All attempts to demonstrate any constant lesion of the nerves or nerve-centres have hitherto failed. The inflammation of the vagus trunk described by Hufeland, Breschet and others has been disproved. The irritation of the branches of the vagus by enlarged or inflamed bronchial glands, advocated by Guenneau de Mussey and Friedleben, has nothing positive to support it, and is on its own merits extremely improbable, considering the frequency with which these changes are met with without any symptoms at all resembling whooping-cough.

3. For the catarrhal theory it is necessary to assume that it is not a common but a specific catarrh, in order to account for its infectiousness. The peculiar attacks are, it is presumed, from their resemblance to those of a foreign body

in the larynx, due to the irritation of the bronchial secretion when it has reached the larynx ; but against these arguments it must be urged that the resemblance to the paroxysms of cough and dyspnœa produced by a foreign body is only remote, and that laryngitis or bronchitis with copious secretion is unattended with any symptoms of the kind. To explain this difficulty it is held that the secretion of whooping-cough is of a peculiarly irritating nature, acquired by the presence in it of specific organisms, which organisms various authors profess to have discovered.

Letzerich [1] in 1870 described fungi with mycelium and spores forming masses large enough to be visible to the naked eye, which commenced to grow about the epiglottis and spread downwards, the paroxysm being produced when they reached the glottis. In 1874 he further described masses of micrococci to which he also attributed specific value, and in both cases he claimed by the injection of the growth into the trachea of rabbits to have produced a characteristic catarrh. These results were confirmed by Tschamer [2] in 1876, but contradicted in all respects by Birch-Hirschfeld [3] in 1878, and by Rossbach [4] in 1880.

Burger [5] in 1883 described a bacillus, and Deichler [6] in 1886 a protozoon ; Afanassijew [7] in 1887 found a small bacillus which in dogs produced bronchitis and pneumonia, with sputum somewhat like that of whooping-cough. These observations Samstchenko [8] confirmed, and found the bacillus not only in the secretion in the trachea and lungs, but also in the liver and spleen.

Ritter [9] described a small diplococcus, occurring in straight or clustered chains, which he was able to cultivate and by which he produced in dogs an affection which resembled whooping-cough.

Cohn and Neumann [10] discovered a streptococcus occurring in long and short chains.

The search for the specific germ of whooping-cough has so far not been successful. The difficulties of isolation are very great, and inoculation experiments upon animals have hitherto proved failures ; for though in dogs catarrh and cough can be produced which resemble whooping-cough, it appears that the same symptoms may be produced by different organisms, so that the test is not conclusive.

On the whole the facts of the disease seem best explained if whooping-cough be regarded as an infectious zymotic disease, the stress of which falls chiefly on the nerve centres in the medulla, but which produces at the same time local manifestations in the air passages in the form of a peculiar or specific catarrh.

A close pathological analogy to whooping-cough seems to be afforded by hydrophobia, a resemblance which may have been present to Trousseau's mind, for in his Clinical Lectures hydrophobia is the subject which he has placed immediately following whooping-cough. If this be an accidental coincidence, it is remarkable.

ETIOLOGY.—Whooping-cough is undoubtedly infectious, and is transmitted through the breath or the sputum directly from the sick to the healthy. It is probably impossible for a third person to carry the infection, unless by wearing clothes upon which the sputum has fallen.

The period of **incubation** is very uncertain, some authors putting it at as little as two days, others at as much as fourteen. In many cases it seems to be about a week. The infectiousness is at its maximum during the convulsive stage, but it is active enough during the initial catarrh, and probably lasts some time after the whoop has ceased, so that patients cannot be considered safe to mix with others till from four to six weeks after the last whoop was heard.

The **protection** afforded by an attack seems almost complete. Second attacks are extremely rare, rarer, indeed, it appears than in almost any other of

[1] *Virch. Archiv*, lvii. 518, and lx. 459.
[2] *Jahrber. f. Kinderheilk*, 1876, N.F., x. 174.
[3] *Clbt. f. Kinderheilk*, 1878, i. 115.
[4] *Berl. klin. Woch.*, 1880, 253.
[5] *Ibid.*, 1883, xx. 17.
[6] *Zeitsch. f. wiss. Zoologie*, 1886.
[7] *St. Petersb. med. Woch.*, 1887, 323.
[8] *St. Petersb. med. Woch.*, 1888, 193.
[9] *Münch. med. Woch.*, Nov. 8, 1892.
[10] *Arch. f. Kinderheilkund*, xvii. 24.

the ordinary zymotic diseases. Two cases each are recorded by Ozanan [1] and Trousseau, and another by Charles West.[2] The last occurred in a girl of 7 years of age who had had the first attack at the age of 3.

Whooping-cough is especially liable to occur in epidemics which spread themselves over many months, but in large towns it is hardly ever completely absent. Hirsch seems to be of opinion that whooping-cough may arise sometimes spontaneously, independently of infection, but this is opposed to modern views.

Its **distribution** is universal. Hardly any part of the world seems free from it. It flourishes alike in hot and cold regions, in high altitudes and in low-lying places. All that can be said is that the tropics are, as a rule, not favourable to its development. Iceland and the Faroë Islands are comparatively free, probably owing to their isolation. Races and nationality are without influence, for all the world seems liable in the same degree.

In regard to season, the outbreak of an epidemic is favoured by winter and spring, but, once started, season has little effect upon its spread, though cold and damp seasons greatly increase the severity and mortality in proportion as they predispose to catarrh.

Its relation to other diseases.—Whooping-cough occasionally follows scarlet fever and small-pox, but then probably only as an accident.

To **measles** it may stand, however, in some closer relation, for the number of instances of this kind is greater than could be explained by mere coincidence. It may be that there is the same tendency to catarrh in each, and that to this catarrh the special susceptibility is due. Usually the whooping-cough follows the measles, and at an interval of from two to eight weeks, but it sometimes precedes and may even run concurrently. When closely following measles the prognosis is rendered less favourable owing to the increased liability to bronchitis and broncho-pneumonia.

In the last century whooping-cough was described as frequently associated with intermittent fever. Recent instances of this are not forthcoming, so that in all probability the fever referred to was really that of broncho-pneumonia.

Age.—Whooping-cough is essentially a disease of childhood, although no age is exempt, but considering that the immunity provided by one attack is almost complete, and that few children escape, it is obvious that cases in the adult would of necessity be rare.

West's statistics, based on 1367 cases, are as follow:—Before the age of 2 years, 42·2 per cent.; between 2 and 3 years, 14·5 per cent.; between 3 and 4, 26·2 per cent.; between 5 and 10 years, 15·5 per cent.; and above the age of 10, 1·6 per cent. In other words, 82·9 per cent. occur before 5 years, and 98·4 per cent. before 10. Macall's statistics yield similar results, viz., 52 per cent. before 2 years, 32 per cent. between 2 and 4, and 8 per cent. between 4 and 6, *i.e.*, 92 per cent. before 6, and 8 per cent. after. Attacks in middle life are very rare. Steffen records one between 40 and 50; Heberden two, one in a woman of 70 and the other in a man of 80. In the Registrar-General's Statistics for the year 1884, quoted below, a fatal case is recorded in a man over 65, and in those for 1887 another in a woman at the age of 75.

Whooping-cough is described as relatively rare in infants before 6 months, probably because they are less exposed to infection, but partly also, perhaps, because the disease is not so easily recognised, the whoop in little babies being by no means so characteristic or common.

Watson describes a case in a new-born infant, the mother having had whooping-cough four weeks before its birth. Bouchut records another which was infected on the second day, and had a characteristic cough on the fifth. Barthez and Rilliet also mention similar cases.

[1] *Hist. Med.*, i. 231.

[2] *Lect. on Children*, 273.

In adults, though cases are rare and the disease less severe, the affection often runs a long course and leaves a good deal of delicacy of the throat and air passages behind. I remember a young lady who had whooping-cough at the age of 25, and whose voice remained weak for nearly two years afterwards, so that she was unable to sing or even to speak for long at a time.

Sex.—Female children are stated to be more liable than male, and also to suffer more severely, but statistics do not seem always to bear this statement out.

The mortality statistics to be quoted later show at any rate that in the five years taken, about 1500 more females died than males, and the proportion of death was that of 16 females to 13 males.

The sickly and weak are not more prone to contract the disease than the robust, but, of course, they suffer more seriously and are more likely to succumb.

Pregnant women are said to be especially susceptible, but the number of cases in adults altogether is so small that this statement is probably an error. What is of more importance is that they do not, as a rule, abort, though from the violence of the cough this might have been anticipated.

It used to be said that animals might be infected from man. Direct experiment does not support this statement, and if it were true, it would be remarkable that there are no instances of it recorded in recent years.

DIAGNOSIS.—When the whoop is present the diagnosis is evident, but in the early stage, before the whoop has appeared, and in the later stage, after it has subsided, there may be nothing to indicate that the affection is anything else than an ordinary catarrh. But even in the early stage, paroxysms of short, explosive coughs might raise a strong presumption in favour of whooping-cough, and this presumption would be confirmed if the attacks ended with vomiting.

In small infants the whoop is often absent, and this may account for the difference of opinion as to the frequency.

Again, with the occurrence of pneumonia or broncho-pneumonia, the whoop disappears, and the true nature of the case is not recognised until the whoop returns during convalescence. This is the explanation of the so-called cure of whooping-cough by acute disease.

From other paroxysmal coughs the history and course of the case, and the character of the cough are sufficient for the diagnosis. Cough due to a foreign body in the larynx is only likely to create difficulty in little infants, or when the body is impacted in the larynx and yet does not produce marked obstruction.

Hysterical coughs are sometimes attended with crowing or whooping, but though the cough may be very frequent or almost continuous, the paroxysms are not severe, and vomiting rarely occurs, besides which the patients are adult, or at any rate about the age of puberty.

PROGNOSIS AND MORTALITY.—The prognosis is, on the whole, good, for in uncomplicated cases recovery, when it takes place, is usually complete.

The general mortality is low, but it differs a good deal in different places and in different epidemics, varying from 2 to 15 per cent.; but, as in influenza, the number of persons attacked during the prevalence of an epidemic is so large, that a perceptible impression is made on the general death-rate. General mortality statistics are, however, of little use in individual cases, and in any given case the prognosis varies a good deal according to the age and general health of the patient, the nature of the complications and other conditions.

Age is by far the most important element in prognosis. Other things being equal, the younger the child the greater is the danger to life, as the following table compiled from the Registrar-General's Reports of the years 1884–8 inclusive shows.

Deaths from whooping-cough at different ages.
(Percentage.)

Totals.	Under 1	1–2	2–3	3–4	4–5	Total. Under 5	5–10	Above 10
6457 Males . .	40·7	32·7	12·4	7·5	3·3	96·66	3·2	0·14
7979 Females .	36·82	32·18	14·8	8·2	4·4	96·18	3·7	0·2

In the statistics of the year 1888, the deaths during the 1st year are thus distributed :—

	First 3 months.	From 3 to 6 months.	From 6 to 12 months.	Total per cent.
Males . .	8·5	11·5	26·8	46·8
Females .	7·8	9·3	24·6	41·7

Of the fatal cases, 40 per cent. occur in children under 1 year of age, more than 70 under 2, and more than 96 per cent. under 5. These figures represent, of course in another way, the fact that whooping-cough is a disease of early childhood, but it is also true that the actual mortality is increased the younger the child is.

Thus Voit's[1] statistics show that under 1 year, 25 per cent. of the infants attacked die; between 1 and 5, however, only 4·8, and between 6 and 15, only 1·1.

The reasons are that infants have but little power to cough and are liable to get collapse, pneumonia and bronchitis, all grave complications in little children; that they lose appetite and will not take food; and that they are so liable to suffer from diarrhœa and vomiting, and to develop marasmus.

Sex.—Under 2 years of age the mortality in the two sexes seems to be equal, but after 2 the mortality among females is rather higher.

The general health.—The more weakly the child the more likely it is to succumb. Strong infants may pull through a severe attack, while a slight attack may prove too much for a weakly child, and it is as factors in reducing the vigour and power of resistance that rickets, syphilis, and chronic diarrhœa are of importance.

The **position in life** also has an influence in a similar way, the risks being less in well-fed, well-housed children, who can be carefully tended and properly fed.

The severity of the attack.—The risks vary with the number and violence of the paroxysms. The paroxysms may be numerous and yet slight, or, on the other hand, few but severe. As a rule, the number and severity vary together. Where violent paroxysms frequently recur, the child is worn out by the fatigue of coughing, and by the want of sleep, as well as by the want of food, for not only is little taken but that little is brought up again by vomiting.

Season.—As one of the chief dangers is bronchitis and the complications it brings with it, season affects prognosis in so far as it predisposes to catarrh.

Voit shows that in Würzburg the mortality in winter is 10 per cent., in spring 7·7, in summer 2·4, and in autumn 2·8.

[1] Hagenbach in *Gerhardt's Kinderkr.*

Bad weather has a further influence in retarding convalescence by confining children within doors, and on this account recovery is more rapid and complete in the summer.

Complications.—Of the complications, *bronchitis* and *broncho-pneumonia* are the most serious, for more than half of all those attacked by them die, and in children under 2, even as many as two out of three. Moreover, of the total mortality from whooping-cough, 50 per cent. is due to one of these affections; and not only are they the most frequent causes of death, but they are the most likely to leave chronic ailments behind, which permanently damage the life.

Next in order of gravity come *convulsions*. Of this complication it is reckoned that about 16 per cent. die, but, as has been already stated, the risk which convulsions brings depends in any given case upon their cause, for where convulsions are due directly to the paroxysm, they are almost always fatal.

The character of the prevailing epidemic.—The fatality of whooping-cough varies greatly in different epidemics in the same place, and also in the same epidemic in different places, but, speaking generally, the prognosis may often be aided in any given case by considering the general character of the prevailing epidemic in that district. In this respect whooping-cough resembles the other common infectious epidemic diseases.

During convalescence the risks are small, but little children are often left feeble and ailing, and it may be many weeks or even months before they recover their previous health.

TREATMENT.—When whooping-cough is epidemic, children should mix as little as possible with other children, children's parties should be especially avoided, or the family might be sent away at once from the infected town into the country. When one child in a family is attacked it should be at once isolated, but, owing to the great infectiousness of whooping-cough in the early stages, isolation is rarely successful.

In many of the mild cases little in the way of treatment is required. It is sufficient to protect the patients from catching cold, to feed them with simple but liberal diet, and administer some tonic. Above all they should be sent as much as possible, without fatigue, into the open air, when the weather is suitable.

The early treatment, if any be required, will be that of a catarrh. The necessity of careful management of the early catarrh of whooping-cough cannot be too strongly insisted upon, for want of care may easily convert what might have been a simple attack into a severe or even fatal one.

Special treatment commences with the paroxysmal cough. The attack may be easily protracted by want of care, but it is very doubtful if there are any means of cutting it short, and the great variation in the character and severity of the disease in different individuals, even of the same family, makes it extremely difficult to judge of the effect of remedies in this respect.

The antidotal or protective agent remains yet to be discovered, and treatment must, for the present, be mainly symptomatic, but much can be done by the treatment of symptoms, and by guarding against complications and dealing properly with them when they arise, to diminish the severity and danger of the attack.

The treatment is greatly determined by the age of the patient, by the number and severity of the paroxysms, by the amount of catarrh, and by the complications which arise. The younger the child the greater the liability to complications, and the greater the danger they bring with them. Babies and children under 3 in all cases, even if there be no catarrh, are best kept within doors during the paroxysmal stage, and if there be catarrh, to one room or

even to bed. Above 5, if the attack be mild, they may be allowed to go out daily when the sun shines and the weather is warm; but in all cases the arms, neck and legs must be properly covered, and the pretty but irrational custom in such a changeable climate as ours of leaving those parts unprotected must be absolutely discarded.

If the patients are confined to one room the temperature should be kept uniform at about 60° F., and provision made to keep the air fresh without a draught. When the children are in bed the face may be covered with a thin shawl, and the window even opened on a fine day with advantage for a few minutes at a time. If the children are well enough to move from the night to a day nursery, they should not change until the day room is warmed up to 60° F.; they should then be well wrapped up with a light shawl thrown over their head, and transferred quickly across the passages. Baths, when required, should be given at night before a warm fire, so that the children may be carried at once from the bath to bed.

As the paroxysms are so easily brought on by excitement or worry, quiet occupation should be provided, and teasing or punishment avoided. Lessons are best given up except so far as they provide light occupation and amusement. Considering the irritability which confinement and sickness causes, there is nothing more trying and wearying to manage and amuse than a whooping-cough nursery.

The diet should be light, nourishing, and easily digestible. Small quantities of food only should be given at a time, for overloading of the stomach will almost infallibly bring on an attack of vomiting and lead to the rejection of the meal.

Where vomiting after food is frequent, the meals should be given immediately after a paroxysm, and they are then often retained. The appetite is nearly always greatly impaired, partly from the confinement to the house, and partly as the result of the disease. The elder children can usually be coaxed with more or less success to take food, but infants often refuse the breast or bottle for hours together. In a very refractory case it may be even necessary to resort to nasal feeding. The loss of appetite, as well as the dyspepsia which develops, is in some measure due to the mucus having been swallowed which has been coughed up from the air-tubes. An emetic is then of great service, for it not only unloads the air-tubes of the mucus but also clears it out from the stomach. In infants, owing to the presence of the mucus, the sugar and starch of the food are extremely prone to ferment and to set up troublesome intestinal as well as gastric dyspepsia, so that the diet should contain as little of these substances as possible.

When vomiting is frequent and severe, a drop or two of opium may be given by the mouth, but not of course to babies, and Drosera (one or two drops of a 1 in 10 tincture) is also said to be of great use.

Treatment of the actual paroxysm.—With infants who cannot help themselves, the moment the first cough is heard they should either be taken up from bed, or have the shoulders raised so as to put them in the best position for effective coughing, and the head should be leant forward so that the mucus and saliva may run easily from the mouth. If the mucus be in large amount, it should be wiped out of the mouth and nose with a thin cloth, so that it may not impede the inspirations which follow. If this be not quickly done the mucus may be sucked into the glottis, greatly increase the dyspnœa and distress, or even lead to suffocation. When the paroxysm is prolonged and the breath long held, so that the face gets very blue, a puff of air blown sharply on the face two or three times will start the inspiration; or a cold wet sponge may

be applied. In older children the face may be freely splashed with water, or the hands plunged into cold water up to the elbow. Older children, if in bed, sit up when the paroxysm begins, or, if out of bed, run to the nurse or to a chair or table and lay their head upon it, or ask to have the head held by the nurse. This they do partly to steady and support the body during the severe paroxysm, partly to fix the shoulder muscles so as to get the breath and to cough with most effect.

An Indian nurse told me that it was a custom among the natives to hold the children up by the hands when the paroxysm commenced, so that the feet were just off the ground, and that this cut the paroxysm short. I have frequently tried this, and think it does somewhat shorten the paroxysm, but it is also certain that the children do not approve of the handling.

As the paroxysm is the most important part of the disease, and that to which many of the grave complications are due, treatment has naturally as its chief object that of reducing its severity and the frequency of its recurrence. Special preference will be given to one or other remedy according to the theory of the disease adopted, but there can be no doubt that the most important and serviceable remedies belong to the sedative or antispasmodic class. Chief among these are belladonna, atropine, hydrocyanic acid, and the bromides, forming, as it were, one group; opium, morphine, chloral, chloroform, and hyoscyamus, forming another; while a third group might be made to embrace antiperiodics, such as quinine and antipyrin.

Belladonna and *atropine* are useful as diminishing spasm, but they do not shorten the disease. They should be given in gradually increasing doses until evidence of physiological action is obtained as shown by dilatation of the pupil. Children bear large doses of belladonna well, and the small doses usually prescribed are insufficient. An eighth of a grain of the extract, or two to four minims of the tincture, may be given as an initial dose to quite young children. Eustace Smith advocates the combination of sulphate of zinc gr. $\frac{1}{6}$ with liquor atropine ℥ $\frac{1}{2}$ in glycerine and water night and morning to begin with, the dose being gradually increased as required.

Hydrocyanic acid was a popular remedy some years ago, but it has gone out of fashion. Dr. Charles West advocated its use in doses of half a drop of the dilute acid of the pharmacopœia to a child nine months old, and so in proportion to older children. According to his statement it exercises in many cases an almost magical effect upon the cough, but it is a drug which must be used with caution, for poisonous effects may be produced, and it is wise to discontinue it if no effect be produced upon the cough in two or three days.

The bromides have their use not only in diminishing the spasm, but in allaying the general irritability of the nervous system which is almost part of the disease. They are most useful when the paroxysms are much increased at night, and a dose given in the evening often secures a fair night's rest. The effect seems to be the same whether the soda, potash or ammonium salt be given, and sometimes hydrobromic acid is equally efficacious.

Bromoform may be administered internally in doses of 2–4 minims thrice daily to a child of a year old. It is best given with syrup and mucilage. It is highly spoken of by some authorities, and is said to reduce the frequency as well as the severity of the paroxysm, but I do not myself consider that its efficacy can be compared with that of belladonna.

Like the bromides, *chloral* and *croton-chloral* have their use, especially when the rest is greatly disturbed.

In very severe and prolonged paroxysms, inhalations of *chloroform* may be sometimes employed with advantage, but this is a remedy that is not free from

risk, and the cases that are fit for it are few and far between. *Ether* has still less to recommend it; *nitrite of amyl* has also been advocated, but its anæsthetic action is more than doubtful, and its influence upon the paroxysm nil.

Hyoscyamus and *conium* are often added to other drugs as sedatives, but are hardly ever prescribed alone.

Opium and *morphine* are, of course, the most important remedies in the sedative group. They are, as already stated, of use in allaying vomiting. They may also diminish spasm and improve sleep, but the greatest care is required in their administration, especially to young children, and they are in all cases contra-indicated where there is much catarrh or secretion in the air-tubes. These drugs, therefore, in whooping-cough, though powerful in effect, are limited in application.

Quinine has been recommended in full doses; for example, 1–1½ grains for a child of 1 year old three or four times a day. Its use is chiefly in the later stages as a general tonic. The same applies to *antipyrin*, which has been given in doses of a grain for each year of the child's age.

Anticatarrhal remedies find their place only where there is much catarrh, but this is, in many cases, too slight to require treatment. If there be much viscid secretion in the tubes, expectorants will be of service, and so will inhalations of benzoin, camphor, or turpentine. It is from their effect upon the bronchitis that the internal administration of benzoin, sulphur, chloride of ammonium, and such-like remedies have acquired their reputation in whooping-cough. In a similar way alum may give relief when the secretion is profuse. Emetics have their use when the tubes are loaded with secretion; but, as already stated, the act of vomiting discharges the mucus from the stomach as well. It is to this double effect upon the bronchi and the digestive tract that Watson's favourite combination of ipecacuanha and rhubarb owes its reputation.

Counter-irritation to the chest, in the form of rubbing with stimulating liniments, is a popular and useful remedy. The liniments commonly used are Lin. Camph. Co., Lin. Terebinth Acet., Lin. Sinapis Co., and Roche's embrocation.

An entirely different line of treatment is suggested by the theory that whooping-cough is a local specific catarrh, dependent upon the growth of specific organisms in the upper air passages. With this view *antiseptic inhalations* and *sprays* have been advocated. The sprays are, however, certainly useless, for it is impossible to get children to apply them properly. Whatever good may appear to result from a spray is due to the internal effect of the drug produced by swallowing the solution. Volatile antiseptics diffused in the air have more to recommend them. It was an old belief that the vapours present in gasworks diminished the severity of the paroxysms, and children were often sent to reside in or near gasworks for that reason. Of recent years the vapours of various coal-tar derivatives have been volatilised into the air of the room; for example, creasote, carbolic acid, cresylene, sulphur, or ammonia, and patients have even been caused to breathe common illuminating gas. In a similar way turpentine or some other pine-oil, or petroleum, have been employed. These latter remedies, placed in a bronchitis kettle and used with steam, have as much effect as in bronchitis and no more. They may be of comfort where there is much catarrh, but in so dilute a form they can have no specific action. To produce any distinct antiseptic effect, they must be used in as concentrated a form as possible, the pure drug being evaporated over a lamp, and the air as nearly as can be saturated with it. In the concentrated form I believe that creasote, and carbolic acid, as well as cresylene, do diminish the severity of the paroxysms; but considering how slightly volatile these substances are, their effect can hardly be due to direct

antiseptic action, for they do not reach the larynx in a sufficiently concentrated form to have any such effect. Mercury was used in former years probably for the same reasons, both internally and by inunction, but its use is now entirely given up.

On the same theory *direct applications to the larynx* have been advocated, either by the brush, sponge, or insufflation of powders. The brush or sponge is practically unavailable for children, and the excitement produced by forcing the treatment against resistance does more harm than the remedy could do good.

The *insufflation of powders* has also little but theory to recommend it. Quinine, boracic acid, iodoform, and others have been used, and successful cases recorded, but this treatment has been so far received with but little favour.

Among the miscellaneous remedies, *Castanea*, *Vesca*, *Clover*, and *Pulsatilla* have been used as sedatives, and are stated to reduce the number and severity of the paroxysms. They may all be given in the form of decoction made with one ounce of the dried leaves to one pint of water. Decoction of clover I have often used with advantage, and it forms at any rate a pleasant vehicle with the addition of some liquorice for the exhibition of other remedies.

If after a severe paroxysm the child fall into a dull lethargic state, and the sensorium show signs of becoming oppressed, steps must be taken to arouse it and to stimulate the nervous system. With this object much good may be done by a hot mustard and water bath, by friction with stimulating liniments, by hot poultices, or by dry cups applied between the shoulders or over the lower parts of the chest.

Complications must be treated as they arise in the usual ways, but they do not call for special consideration in this place.

During convalescence, tonic treatment is in all cases necessary. Quinine, iron, cod-liver oil, maltine, and liberal diet are the common and best remedies. As soon as the child is well enough, the weather permitting, it should be sent out as much as possible into the fresh air and sun. Small children, and especially babies, sometimes seem steadily to refuse to improve until they are sent away from home, when they immediately rally, and that without any other fresh treatment. Indeed, in all cases convalescence is greatly accelerated by change to the seaside or to some bracing place in the hills.

54. HYSTERICAL NEUROSES.

Of the alteration in the rhythm and rate of breathing met with in emotional states, *e.g.*, sighing, sobbing, laughter, and some forms of cough, nothing need be added to what has already been said in other places.

In hysteria these and similar conditions are observed.

Hysterical Asthma is the most striking of these, but would be more appropriately described as *Tachypnœa* rather than asthma, for there is no dyspnœa; and the respirations are not asthmatic in type at all.

Hysterical Paralysis of the Respiratory Muscles is very rare, but two remarkable conditions are referred to in the appropriate places, viz., hysterical abductor paralysis and hysterical paralysis of the diaphragm.

55. PERIODIC RESPIRATION.

The respirations in health are remarkably regular both in depth and frequency, and they remain so under many pathological conditions, even when they are greatly quickened as in fever, or rapid and shallow as in pneumonia.

When the respirations become irregular, they may vary both in depth and in frequency. This is familiar under emotional conditions, *e.g.*, in laughing, crying or sobbing. Where there is dyspnœa of moderate degrees, there are irregularities of a different kind; the respiration may for a time be regular, then, as the need of air makes itself more felt, they become deeper and fuller, to become again as they were at first as soon as the discomfort is relieved. A similar kind of irregularity is met with when the stomach, or abdomen generally, is distended, as is seen in the occasional sighing, yawning, or deep breathing of flatulent dyspepsia, etc.

All these irregularities of breathing are themselves of irregular recurrence.

There is, however, one class of irregular respiration in which the irregularities are periodic. The respirations occur in groups which recur at regular intervals, each group having the same general characters. This is called *rhythmical* or better *periodic*, respiration, for the normal movements of respiration are rhythmical.

Of periodic respiration the most striking and characteristic example is that known as Cheyne-Stokes breathing, of which many of the other varieties of periodic respiration described are but modifications, for they occur under similar clinical conditions and have the same clinical significance.

56. CHEYNE-STOKES BREATHING.

Cheyne-Stokes Breathing is especially characterised by the long pauses, or periods, during which the chest is motionless and respiration stopped. At the end of such a pause the respirations begin, being at first shallow, but rapidly increasing in depth, until the respiratory excursion reaches almost its maximum, when they become less and less deep, until they end again in the pause, during which all respiratory movement ceases, it may be for many seconds, perhaps even half a minute or more. These respiratory phases, with their regular and constantly recurring crescendo and diminuendo, separated from each other by periods of respiratory still-stand, form a striking clinical picture, of which the best idea will be given by the description of a typical case such as that which follows, and by the diagrams accompanying it.

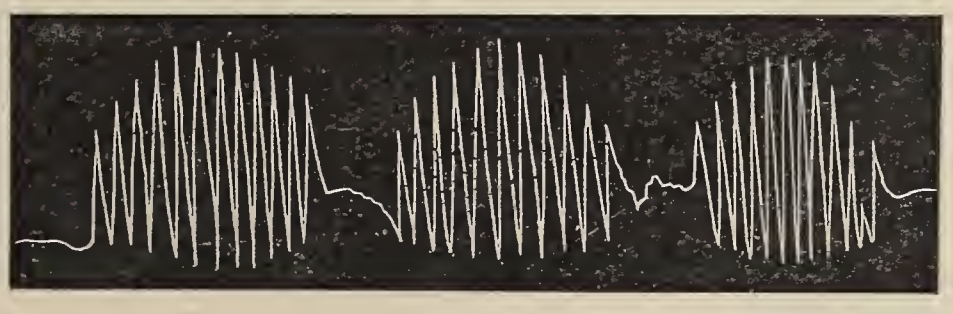


Fig. 141.

A semi-diagrammatic tracing of Cheyne-Stokes breathing. The upstrokes represent inspiration, the downstrokes expiration; the more or less horizontal line indicates the pause.

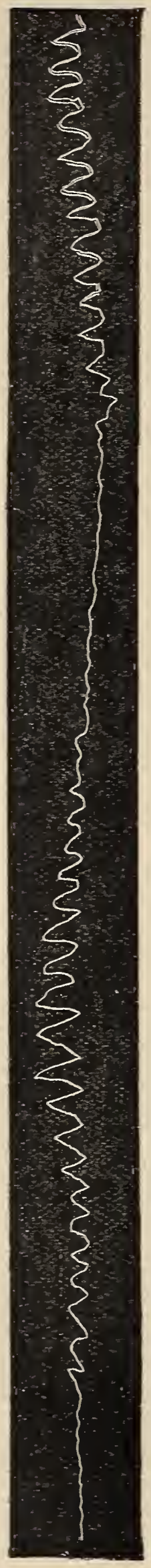


Fig. 142.—Tracing of Cheyne-Stokes breathing, obtained from the case described.

Case of Cheyne-Stokes Breathing of at least three months' duration in the course of granular kidney.—The patient, a man of 55, had been in good health, except for occasional attacks of gout, until about twenty months before his death. He then noticed that his breathing was becoming short on exertion. This gradually grew worse. At the end of the year he had a very severe paroxysm of dyspnœa, which came on quite suddenly and without cause, while he was in bed. It lasted half an hour. He suffered no pain, but was in intense distress, and felt as if he were going to die. He had three attacks of a similar character, the last and most severe one occurring three weeks before he came under observation. From that attack he never recovered, the breath continuing so short that it was difficult for him to walk even a few yards, but he had never kept his bed entirely.

The patient was a well-developed man, but his muscles felt flabby, and his complexion was sallow and earthy.

The radial and temporal arteries were tortuous and thickened, and the tension high; pulse 104, regular; the respirations 48, shallow, chiefly diaphragmatic, and expiration prolonged.

The heart's apex was in its normal place, and the cardiac dulness was not increased. The sounds were normal, except that the second was accentuated at the apex.

The urine contained some albumen, about $\frac{1}{16}$, and had a sp. gr. of 1010.

During an attack of dyspnœa he preferred to sit upright in a chair; but at other times lay most comfortably on the back.

The attacks were evidently cardiac in origin, the heart being probably fatty; the cause of the heart affection was granular kidney. The retinæ were healthy.

The patient was ordered three minims of tincture of strophanthus and five minims of tincture of nux vomica, with calumba and soda mixture every five hours; he was also ordered brandy.

October 4.—The patient had had but little sleep; he had been restless and wandering, talking incoherently at times, and wishing to get up, under the impression that he was at his business. The pulse was slower, but of very high tension. The respirations 30, but not so shallow. The eyes had a staring and wondering look, and he took some time to comprehend what was said to him.

October 5.—The breathing had been bad the whole night, and though there had been no actual attack of dyspnœa, still the breath had been short enough to keep him awake, and he had hardly slept at all.

This morning there was well-marked Cheyne-Stokes breathing. The respirations were about 48 in the minute; the cycles consisted of 30 to 35 respirations, and then a pause lasting about twenty seconds. During the pause the pulse did not change in character or rate, nor did the pupils, nor was there any alteration in the colour or appearance of the patient, who seemed quite unconscious that anything unusual was occurring.

October 8.—Patient had been in the same condition; though constantly drowsy he seemed never to sleep, but dozed for a few minutes at a time night and day. Last night he fell asleep for about ten minutes, and then woke up in a fright with his hands and face bathed in a cold clammy sweat.

The urine was examined again, and contained some granular casts. The area of cardiac dulness was slightly increased upwards and to the left. Five minims of tincture of digitalis were substituted for the strophanthus.

On October 10 a small dose of morphine was given *sub cutem*, and he slept all night. He was sick several times the next morning, but the Cheyne-Stokes breathing was not so marked.

On the 13th a systolic murmur was detected at the right base, but it was not conducted into the carotids.

On the 17th the pauses in the respiration were absent, and remained absent for a week.

On the 24th the respirations were 40, but easy, and though there was no distinct Cheyne-Stokes breathing, still there were every now and then intermissions, from two to four respirations being missed.

The pulse continued of very high tension, and was at times slightly irregular. The heart, which had been slowly dilating, now presented its apex in the sixth space, an inch and a half outside the nipple, and the dulness reached above the fourth rib. At the apex, and limited to it, there was a short harsh systolic murmur; the base-sounds were feeble, and the accentuation of the second aortic sound had disappeared. The patient, however, said he felt better.

On the 25th the apex murmur was heard in the mid-maxilla.

On October 31st the heart was in much the same condition, but at the right base the first sound was weak and the second somewhat ringing, and neither the systolic nor the diastolic interval was quite clear, though there was no distinct murmur.

The short pauses previously described, or rather intermissions of three or four respirations, continued. The pulse remained of the same high tension.

On November 2nd well-marked Cheyne-Stokes breathing returned after having been absent from October 17th.

The next day the pauses were absent, but the crescendo and diminuendo in the respiration continued well marked. On the day following these two were gone, but every now and then the patient took a deep sighing inspiration. When asleep the pauses returned, and lasted about eighteen seconds; but on the next day again this was reversed, the pauses being absent when the patient was asleep, and present only when he was awake.

On the 7th the Cheyne-Stokes breathing returned.

On the 9th the patient insisted upon going out to transact some necessary business, much against my advice, and in the evening was very ill with continuous dyspnœa, which culminated during the night in three very severe paroxysms, for which the house physician was summoned. By means of ether and brandy the attacks were relieved.

From this time the Cheyne-Stokes breathing continued until the patient's death, the pauses lasting on the average about twenty seconds, and the whole recurring about once a minute, the number of respirations averaging 40 in the minute.

On the 16th the patient was very restless and semi-delirious, the face greyish livid. The cycle of respiration lasted about a minute, the pause occupying about sixteen to eighteen seconds of this period, and the total number of respirations were about 40; the pulse about 100, not quite regular, and with a feeble wave, but the tension was still above normal. No variation could be detected in the pulse or pupil in the two phases of respiration.

The cardiac dulness was much as before, but the apex murmur could be heard, it was thought, faintly behind and at the right base; both sounds were ringing, and there was a doubtful systolic murmur.

The patient was extremely drowsy, and had to be roused to take food, which he ate with relish, but he fell asleep again at once.

When the patient was told to breathe during one of the pauses, he would make an attempt, but with hardly any effect, for the diaphragm did not respond, nor did the ordinary muscles of respiration; all that the patient could do was to set some of the extra respiratory muscles to work. The contrast between the voluntary attempts at respiration during the pause and the involuntary respirations after it was very remarkable. It had been observed lately that the pause was generally terminated by a short cough, of which the patient did not seem to be conscious.

November 26.—The patient had been getting gradually worse for the last few days. Last night he was so restless that a little opium was given him, after which he slept for about two hours. It did not seem to suit him, for on awaking he was delirious and not so well. The hands were found to be a little swollen to-day for the first time, but the feet were not.

The pauses in the respiration lasted on the average thirty seconds, sometimes longer. During them the patient seemed semi-conscious, the eyes were partly closed, the mouth wide open, and the saliva dribbling from it. The pulse seemed to gain force and volume towards the end of the pause, and to decrease again when the respirations recommenced. Cough marked the end of the pause, and was more marked when the patient was lying back.

The urine was passed unconsciously, and though the patient answered when spoken to, he did not seem able to express his wishes. For a little more than a week from this date the patient developed a ravenous, almost insatiable appetite.

On December 12th the right eye was examined to see if any change in the calibre of the vessels or in the circulation could be detected during the phases, but no change occurred.

Nothing further transpired until the patient's death, except that the pauses became slightly longer and the arterial tension lower. As the tension fell it was observed that the pulse became fuller and stronger at the end of the pause.

The patient died quietly on December 27th, having passed no urine for the last eighteen hours of life. A few hours before death he vomited a quantity of blood-stained fluid without effort or distress.

No *post-mortem* examination was permitted, but there can be no doubt that the case was one of gradual failure of a hypertrophied heart in the course of granular kidney.

It is interesting to note the changes in the respirations which were observed.

1. Cheyne-Stokes breathing was well marked for seventeen days after admission; it then disappeared for a fortnight, to last on its return till the patient's death, eight weeks later.
2. The pauses varied from eighteen to thirty-five seconds; they were longest just before death. At one time the waves of respiration were distinct, though the pauses were absent. At another time during the fortnight's interval the respirations were regular in depth and frequency, but there were every now and then intermissions, from two to five respirations being missed. At another time the place of the intermissions was taken by a long, deep, sighing inspiration. There can be little doubt that these were all but different stages of the same condition.
3. Even during the longest pauses, except just at the last, there was no change to be detected in the pulse, pupil, or appearance, no cyanosis developed, and the patient seemed quite unconscious that he was not breathing; indeed, he appeared to have lost for the time all voluntary control over his respiration.
4. Another important feature in the case is the length of time that Cheyne-Stokes breathing was observed. It was well marked for the first seventeen days, and though absent as such for the next fortnight, still there were irregularities which were clearly of the same character, after which it returned and lasted till death, eight weeks later, so that it is known to have been present for three months certainly, and probably longer, for there can be little doubt that it was present some time before the patient came under observation.

As the result of the discussion which took place upon this paper, several interesting communications appeared in the *Lancet* during the months of March and April 1890, in which a few instances of recovery after Cheyne-Stokes

breathing were recorded, and a few of long duration. The most remarkable was that described by H. S. (*Lancet*, March 27, 1890), of a gentleman aged 92 years, who had presented the phenomenon in a slight degree for many years.

As will be seen, it may fairly be questioned whether some of these cases really belong to the group of Cheyne-Stokes breathing at all.

Associated Phenomena.—Various phenomena have been associated with the different phases of Cheyne-Stokes breathing, *e.g.*, changes in the rate, tension, and character of the pulse, alteration in the pupils, variations in the dyspnœa and cyanosis, etc.

All such phenomena were specially observed to be absent in the preceding very typical case, as in many other recorded cases. Thus we may safely conclude that none of these phenomena are essential, and cannot, as so many authors have assumed, be in any way the cause of the periodic respiration. But as these phenomena are present in some cases it will be well to consider them further.

Pulse rate and tension.—The conditions of the pulse are very variable, and not constant even in the same case.

The pulse rate is sometimes slower during the pause, sometimes accelerated. The beats may even be absent at the wrist, either during the pause or during the respiratory phase.

The volume and tension often undergo no change whatever, but sometimes the volume becomes larger and the tension lower during the pause. In the case described no change whatever occurred in the pulse until shortly before death, and then the pulse became fuller and stronger towards the end of the pause.

Many of these variations in the pulse are simply such as are met with in any case of cardiac failure independent of Cheyne-Stokes breathing. Moreover, irregular variations in the tension and volume of the pulse are not uncommon in the later stages of granular kidney, in the course of which disease so many of these instances of Cheyne-Stokes breathing occur. In connection possibly with the circulation-disturbances may be placed the alternate recession and protrusion of the fontanelles observed in some cases in infants.

Cyanosis and dyspnœa.—The complexion is often markedly pale and not in any way dusky.

Cyanosis and dyspnœa are often entirely absent, and if present could hardly, and do not as a matter of fact, show any marked difference in the different phases.

The dyspnœa which is occasionally present is only such as depends upon the failure of the heart.

It often happens that, though the breathing is obviously short, the patient has no sense of dyspnœa, *i.e.*, no respiratory distress. Indeed, it is remarkable that the patients after a long pause seem quite unconscious of the fact that they have not been breathing as usual and commence breathing again as if nothing had occurred, and without even so much change as a healthy person would show who had held the breath for half a minute or more.

Nor do they complain of their breathing at all, or at any rate more when Cheyne-Stokes breathing is present than when it is absent.

The Eyes.—The eyes may be open or closed, the pupils contracted or dilated. If there be any change at all, the eyes usually close and the pupils contract during the pause. In other words, these patients seem to go to sleep and the eye-changes usual in sleep occur.

If Cheyne-Stokes breathing be present only when the patients are awake, the eyes remain open and the pupils dilated, and if it occur only during sleep, the pupils remain contracted; in neither case do they vary in relation to the phases.

The Mental Condition varies with the original disease. Thus when there is no unconsciousness it often remains clear though somewhat dulled, much as if the patient were a little drowsy, and this becomes more marked during the pause.

When the patient is unconscious, the unconsciousness seems sometimes to be less during the respiratory phase than during the pause, as if the respiratory phase acted like a stimulus to the brain.

Movements.—Whatever movements of the body take place occur during the pause; but, as a rule, the patient lies quite still.

Respiratory phenomena other than those of the periodic respirations are absent. The pause often ends with a slight cough. That a cough is possible shows, as is the fact, that the chest during the pause is in a condition of medium expansion, *i.e.*, in the position of natural rest or equilibrium. In most cases the respiratory phase commences with an expiration, as, indeed, it generally does after the breath has been intentionally held for some time.

These associated phenomena, then, being not constant in all cases alike, or, indeed, in the same case at different times, cannot be in any way the cause of Cheyne-Stokes breathing. They are not essential but accidental, and are the result of the same morbid conditions to which the Cheyne-Stokes breathing itself is due. It follows, therefore, that all explanations of Cheyne-Stokes breathing which presuppose any of these phenomena must fall to the ground.

The various conditions under which Cheyne-Stokes breathing has been observed are very various.[1]

Nervous Diseases.—Meningitis, encephalitis, cerebral hæmorrhage, embolism and thrombosis, cerebellar hæmorrhage, pressure on the medulla and pons by a tumour, an aneurysm or extravasated blood, sunstroke, insanity, and hysteria.

Cardio-vascular affections.—Fatty degeneration of the heart, valvular disease, myocarditis, pericarditis, thoracic and other aneurysms.

General arterial degeneration (*i.e.*, atheroma or granular kidney), hæmophilia, hæmorrhage after severe operations.

Respiratory affections.—Pneumonia, broncho-pneumonia and phthisis, and after tracheotomy.

Miscellaneous affections.—Fatty degeneration of the diaphragm, narrowing of the foramen jugulare but always in association with cardiac or renal disease, severe diarrhœa.

General diseases.—Mostly fevers, *e.g.*, typhoid fever, small-pox, diphtheria, but also cholera, whooping-cough, etc.

After the use of certain drugs, e.g., bromide of potash, chloral, and morphine, the symptoms passing off when the drug was suspended.

It would seem to have occurred sometimes, but very rarely, in *healthy persons* during sleep, after great fatigue, or in advanced life.

In some rare instances periodic respiration is said to be a personal or family peculiarity.

In animals periodic respiration has been observed during sleep, after prolonged exertion, and during hibernation.

During experiments it has been observed after the administration of certain drugs, *e.g.*, chloral, morphine, morphine followed by ether, ether and picrotoxin, muscarine or chloroform, picrotoxin, digitaline, strychnine, sulphuretted hydrogen, urea, kreatin, and carbonate of ammonia; after considerable changes in external temperature; after prolonged immersion, in amphibia; after bleeding, removal of the heart, alternate compression and release of the carotid and vertebral arteries; after section of the medulla, and after various injuries to the brain and medulla.

Such a list is a mere enumeration of the conditions under which Cheyne-Stokes breathing, or some form of periodic respiration resembling it, has been observed. It is obvious that such a list throws no light upon the essential condition which underlies the phenomenon.

Cheyne-Stokes breathing is a pathological and clinical condition, about which we know so little that it is more likely to lead wrong than right if there be included in our consideration the various conditions under which periodic respiration is met with in the healthy man or animal, or in animals under abnormal conditions. These conditions, allied possibly, but not necessarily the same, are of great interest as illustrations, and will, no doubt, prove of importance ultimately in solving some of the difficult questions that arise, but it will be well to place them all aside for the present, and deal only with the pathological and clinical conditions under which Cheyne-Stokes breathing is seen.

The cases fall into two great groups—(1) That in which there is heart-failure of some kind; (2) that in which there is some disease or morbid state of the central nervous system. That is to say, when Cheyne-Stokes breathing is present, the patient is suffering from some affection which places it in one or other of these groups; yet, on the other hand, it is not a necessary accompaniment of any affection in either group, and is in each far more often absent than it is present.

[1] Gibson, *Edin. Med. Jour.*, vols. xxxiv., xxxvi. and xxxvii.

It must, therefore, depend upon some accidental condition, which, however, is much more likely to arise in connection with these two groups of affections than with any other.

These two groups further present certain points of contrast which justify the division.

In the first—that of heart-failure—unconsciousness is the exception, and the other associated phenomena are rarely present, except such as are directly due to the condition of the heart. The Cheyne-Stokes breathing is often the most pronounced symptom, while its significance is of the greatest gravity, for once present in such a case, even to a slight degree only, the patient rarely, if ever, recovers, or lives long. Even if life be prolonged for some weeks, the course of the case is steadily downhill to the end.

In the second group unconsciousness is the rule, and the other associated phenomena are frequently present. The Cheyne-Stokes breathing is overshadowed by other more serious symptoms, among which it is one of the latest to develop, so that it has not the same significance. The prognosis is that of the original disease, which is itself usually of a fatal character, but if it should not prove fatal, the Cheyne-Stokes breathing may disappear.

Thus cases of recovery are recorded after cerebral hæmorrhage and cerebral softening, hydrocephalus and epilepsy, after influenza, after puerperal septicæmia, after uræmia, and after poisoning with morphine.[1]

Cheyne-Stokes breathing is not necessarily continuous; it may intermit and vary as in the case described.

Diagnosis.—The regularly recurring pauses, followed by the rapid crescendo and diminuendo of respiration, are so characteristic, that well-marked cases of Cheyne-Stokes breathing are quite easy to recognise, but this is not so with its different modifications. Yet it is very important to be familiar with these variations, because, by recognising their true nature and significance, much information as to prognosis is obtained, and serious mistakes may be avoided.

The following variations are met with, many of which were present in the case described:—

1. The pauses may be very short, or even absent altogether, and still the crescendo and diminuendo of the respiration be well marked.
2. The breathing may be regular and accelerated only a little or not at all; but there may be at regular intervals intermissions of two or three respirations, or in their place there may be a long-drawn sigh or two.
3. At other times the respirations may be irregular but not periodic.
4. The pauses may be present only at certain times, especially during sleep; but, on the contrary, they may occur only when the patient is awake.

Theory.—There are two parts of Cheyne-Stokes breathing which require explanation—(1) The pauses, and (2) the crescendo and diminuendo of respiration; and it does not necessarily follow that one explanation will do for both.

It is generally agreed that the respiratory centre is at fault, but there is no agreement as to why and how it is affected in so peculiar a way.

The respiratory movements, during the time they are present, do not differ essentially from the normal. They occur with almost rhythmical regularity, though with somewhat increased frequency; and the crescendo and diminuendo is almost entirely in respect of depth of breathing, *i.e.*, in extent of movement. It would seem, therefore, that the respiratory centre was performing its usual

[1] *Cf.* Gibson, *l.c.*

functions, though in a somewhat unusual way. The gradually increased and then gradually diminished activity of the centre, and the periodic recurrence of the phases, are usually connected with its nutrition, and referred to variations in the quality or quantity of the blood supplied to it (Traube); and that whether preceded or not by some change in the general vasomotor condition (Filehne, Broadbent, &c.).

All such theories only put the difficulty a step further back, for they leave unexplained why these rhythmical changes in nutrition or vasomotor action occur.

Some of these theories, for instance those which presuppose vasomotor change, whether of contraction or dilatation, are at once put out of court by the fact that the antecedent conditions given are not constant; neither in all cases nor even in the same case at different times.

If changes in the quantity or quality of the blood cannot explain the phenomenon, the cause might lie in the centre itself (Rosenbach, Luciani).

If the respiratory centre were itself normal, given the pauses, we might, it would seem, anticipate changes in the breathing after the pause, something like those which do occur, and *vice versâ*. The breathing which follows the pause is not unlike that which occurs when the breath has been held intentionally for some time. The respirations then usually commence with an expiration, and continue deep until the want of breath is satisfied, when they diminish again.

On the other hand, given the respiratory phase, with its wide excursion and deep breaths, the pause might be compared with the condition of apnœa, produced by active artificial respiration. This analogy is closer when it is borne in mind that cyanosis is no essential part of Cheyne-Stokes breathing, indeed, it is often conspicuously absent, and that in most cases there is no sensible dyspnœa.

Lesions of the medulla, involving the respiratory centre, cause various changes in the respiration, but rarely, if ever, those of Cheyne-Stokes breathing.

The conclusion, therefore, seems warranted that the respiratory centre in the medulla itself is doing its work fairly well, but that it is interfered with in some way, so that the fault would lie, not in the respiratory centre in the medulla, but elsewhere.

The respiratory centre is no doubt automatic, but at the same time there is no other automatic centre so largely controlled and easily influenced by centres above it. This must of necessity be so, considering the many functions which that centre has to subserve besides that of breathing, especially in man, in connection with the voice and speech; the prime cause of Cheyne-Stokes breathing is then to be sought, it would seem, not in the medulla at all, but in centres above it, probably in the brain.

It is a step in advance, I think, to remove the probable seat of disturbance from the medulla to the nerve centres above it, but, of course, it does not explain why this disturbance is periodic, and occurs at such regular intervals.

Rhythmic action is a property of many living tissues, perhaps of all. Physiological action is not to be represented by a dotted line, in which the dots or dashes represent action and the spaces rest, but rather by a continuous wavy line. There are alternate periods of activity and rest—waves they may be called, very short in some instances, as in the heart action, somewhat longer, as with the respirations, but unexplained as yet in all cases alike. The longest wave of all is that represented by sleeping and waking, and this is ultimately to be referred to the alternation of day and night.

It is conceivable that this great wave, which passes over the body daily, may set up many secondary waves, some large, some small. The little ripples, it may be, are seen in the rhythm of the heart. But larger waves of periodic action will probably be found if looked for. It is such waves as those that may explain the occurrence of Cheyne-Stokes breathing, waves which are controlled or compensated by the higher nerve centres so long as these centres are in a healthy state, but which become manifest or even exaggerated and magnified when the higher centres fail and lose control. The periodicity would then be a phenomenon, not new or unknown in health, but one normally present, though concealed.

If, then, Cheyne-Stokes breathing be due to a loss of cerebral control, which permits normal periodic waves to make themselves manifest, a general principle is obtained which links together the very diverse conditions under which this peculiar periodic respiration occurs. An easy explanation is thus provided of the whole nervous group, while the cardiac group would be explained by impaired nutrition of the brain consequent on the fresh circulation, and in a similar way would be explained the occurrence of Cheyne-Stokes breathing in the course of certain general diseases. Lastly, its occurrence and even long duration in conditions of apparently perfect health would be intelligible.

General speculation of this kind does not, however, in any way diminish the clinical importance of the symptom under the circumstances under which it ordinarily arises. Whatever theory may be adopted, the clinical significance of Cheyne-Stokes breathing is the same. It is of grave import, and, with but few exceptions, means death either imminent or not long deferred.

57. AFFECTIONS OF THE PLEURA.

INTRODUCTORY.

The pleura is a thin serous membrane which lines the cavity of the thorax and is reflected over the viscera within. It thus forms a sac on each side and encloses a cavity—the pleural cavity.

The interior of this sac is lined with flattened epithelial cells, which give it its smooth polished surface.

Everywhere beneath the connective tissue lies a rich plexus of lymphatics, continuous with the lymphatics of the adjacent parts, *i.e.*, with those of the walls of the thorax, the mediastinum, the diaphragm and the lung. These lymphatics communicate with the interior of the sac by small pores or stomata.

Though the surfaces which face each other are moist, the cavity contains no fluid; yet it is probable that there is a continuous circulation of lymph through the cavity, the fluid being removed as rapidly as it enters. This is effected by the respiratory movements, which, by means of the stomata and of the valves in the large lymphatics, act like a pump, and thus keep the pleural cavity dry, *i.e.*, empty of fluid.

INTRA-PLEURAL TENSION.—Under normal conditions the lungs within the chest are in a constant state of tension. They are on the stretch and tend to contract by virtue of their own elasticity. Thus the two layers of the pleura, which in health are in close contact, are subject to a constant strain which tends to pull them apart.

This is called the intra-pleural tension. It is for all practical purposes equal in amount to the elastic contractility of the lung, but opposite in direction, and thus the elastic tension of the lung is positive but the pleural tension negative.

Whether, under normal conditions where the two layers of the pleura are in close apposition, there is a force existing between them (viz., that of a cohesion, as I suggested some years ago)

which balances the elastic contractility of the lung or not, is a question which admits of discussion. *A priori* it is not improbable, and I have shown by experiment that it is possible. However this may be, as soon as the two layers of the pleura are separated, cohesion, if it existed as an active force when they were in close apposition, becomes eliminated, and the lungs tend to collapse.

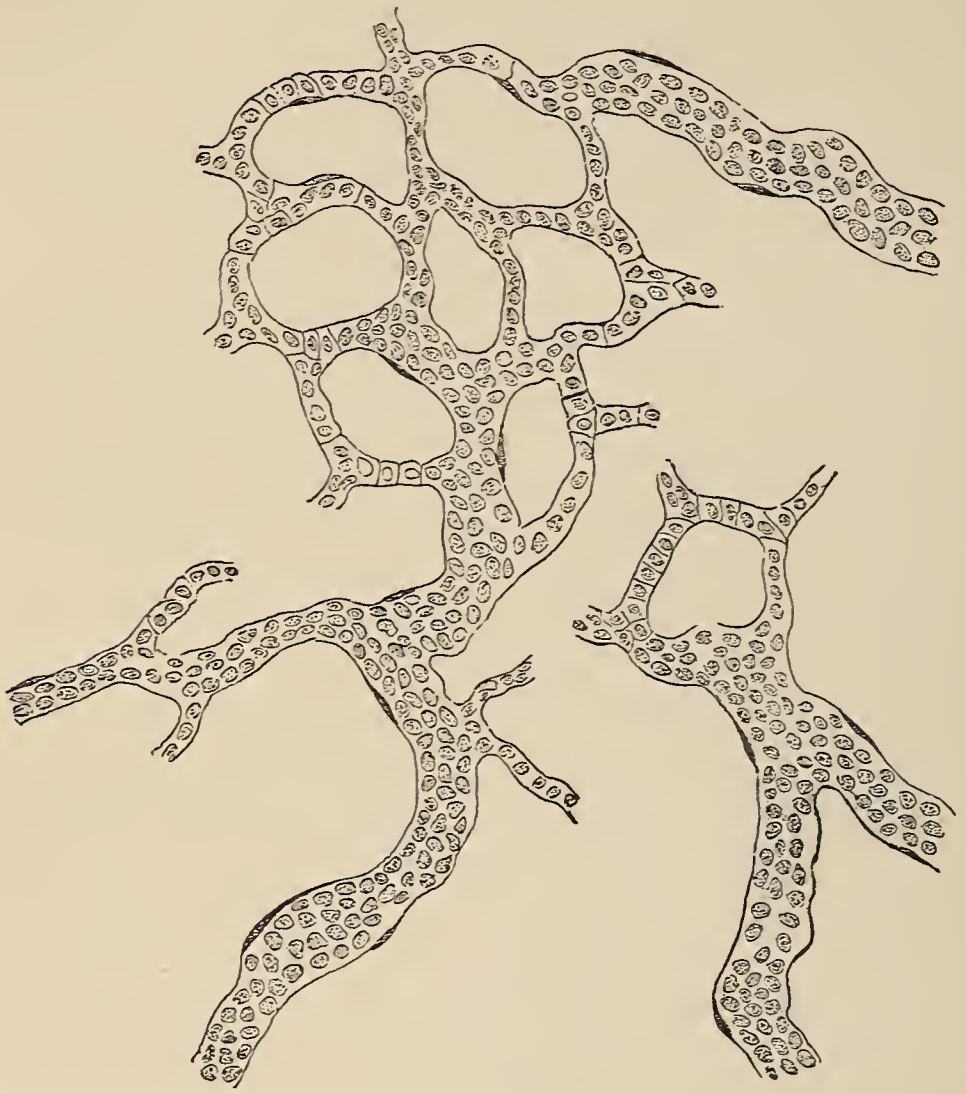


Fig. 143.

Lymphatics of the pleura of the rabbit. Surface view of the lung, showing the superficial network. (From Klein's *Lymphatics*, vol. ii. pl. ii. fig. 7.) *Cf.* figs. on pp. 7 and 8.

There is an interesting specimen in the museum of St. Bartholomew's Hospital (No. 1678*a*), in which the subpleural lymphatics of the lung are infiltrated with cancer, so that the surface of the lung, over a considerable area, is covered with a meshwork of distended lymphatic vessels. The specimen was taken from the body of a patient who died of cancer of the breast, but there were no metastatic growths in the lung.

The factors which combine to produce the intra-pleural tension are, first and foremost, the elasticity of the lungs; secondly, the resistance of the chest walls.

These two factors, though varying considerably no doubt in different individuals, or even in the same individual under different conditions, are constant in any given individual under the ordinary conditions, and are much the same after death as during life.

Besides these there is a third factor, but it is one which is constantly varying, viz., the movements of respiration; for it is evident that on deep inspiration the lungs are more on the stretch and the pleural tension therefore greater, while on expiration the lungs are less on the stretch and the intra-pleural tension therefore smaller.

If the air were stationary in the air-tubes, as it is after death, the pressure in them would be that of the atmosphere; but the movements of the air in and out, during inspiration and expiration respectively, cause slight variations in pressure. Thus Landois and Stirling state that during inspiration the pressure in the lungs falls by a millimetre of mercury, *i.e.*, $\frac{1}{2}$ an inch of water, and during expiration rises by 1, 2 or 3 millimetres of mercury, *i.e.*, $\frac{1}{2}$ to $1\frac{1}{2}$ inches of water. Thus the respiratory oscillation during ordinary respiration would be from $1\frac{1}{2}$ to 2 inches of water.

The condition of the tubes, *i.e.*, whether there be obstruction in them or not, is very important to bear in mind in determining either the elasticity of the lung or the pressure within the pleura; for wherever there is any obstruction in them the elasticity of the lung will be *pro tanto* prevented from coming into play and the true results be not obtained.

If the forces of respiration be eliminated, as after death, then under the normal conditions the intra-pleural tension must be a negative quality, *i.e.*, less than the atmospheric pressure, and less by as much as is represented by the contractility of the lungs.

During respiration also, so long as the breathing is quiet, *i.e.*, not forced or violent, the intra-pleural tension must still be, both on inspiration and expiration, a negative quantity; for, assuming the value of the elasticity of the lung to be that which Donders estimated, viz., 6 to 7 millimetres of mercury, and that the respiratory oscillations are, as stated above, from 1 to 2 or 3 millimetres of mercury, there remain still 3 or 4 millimetres of mercury below which the tension will not fall.

Where the respirations are forced, that is to say, where inspiration is deep or expiration excessive, the conditions may possibly vary considerably. With deep inspiration it must, however, always be negative, and the larger in amount the deeper the inspiration is.

With forced expiration, as in straining, coughing or vomiting, the pressure may be raised even to the extent of 3 or 4 inches of mercury (*i.e.*, 75 to 100 millimetres), but so long as the two layers of the pleura are in close contact with the chest wall, this excessive pressure is supported by the chest walls and does not fall directly on the pleura. On the other hand, under pathological conditions, where the two layers of the pleura are separated, any changes of pressure of the air within the lung are at once transmitted to the contents of the pleura ; but the effect varies greatly, according as the contents of the pleura are practically incompressible, as in the case of fluid, or easily compressible, as in the case of air.

The methods by which the intra-pleural tension has been estimated in health are two :

1. By means of a trocar introduced into the pleura through the chest walls.
2. By means of a tube fastened into the trachea, the pleural cavity being then opened by an incision through the chest walls, first on the one side and then on the other.

In either case the reading is made upon a mercury- or water-manometer connected with the trocar or the tracheal tube respectively.

In the second method, as the elastic tension in the lung is equal in amount to the tension in the pleura, but opposite in direction, the sign must be changed. Thus the positive readings showing the tension of the lung become negative readings in respect of the tension in the pleura.

For man both these methods of investigation are available after death, but the former only during life or under pathological conditions.

Intra-pleural tension is sometimes spoken of as intra-pleural pressure, and under pathological conditions the pressure is sometimes positive and sometimes negative. A certain amount of confusion is thus introduced both in thought and in expression.

This confusion will be removed if it be remembered that the records are expressed in manometer readings ; thus, if the column of liquid in the manometer tube on connecting it with the pleura remains stationary, the reading will be zero, although in reality, of course, the actual pressure within the pleura is that of the atmosphere, and being equal to that within the air tubes might be called one ; but as this is the zero point upon the manometer, it is more conveniently called "zero."

One, two, three, etc., millimetres of mercury or inches of water would mean one, two, or three above or below the atmospheric pressure, as the case may be, and would be indicated by its appropriate sign of "plus" or "minus."

The readings are given in different investigations, sometimes in millimetres of mercury, sometimes in millimetres or inches of water. It is more convenient to use water, because the variations are greater, and therefore more obvious, and in the experiments which I have to record I have measured it in inches ; but the conversion is easily made from one scale to the other ; thus one millimetre of mercury is approximately equal to half an inch or 12½ millimetres of water, and one inch of water is equal to 25 millimetres of water.

The intra-pleural tension as estimated by Donders was equal to 7 or 8 millimetres of mercury, and this is the value usually given in text books.

Perl's experiments, conducted according to the second method, give a somewhat lower value, about 6 millimetres of mercury (*Deutsches Archiv f. klin. Med.*, 1869, vi. 1–5).

As Perl's experiments are the most interesting and important from my point of view, it seems to me desirable to give a short account of the results obtained by him, but in doing this I shall add various comments of my own.

Perl's experiments were conducted with a view to determine the pressure relations within the thorax in different diseases.

They were conducted in the following way :—

A tube was fixed air-tight into the trachea, connected with a manometer ; the right pleura was then laid freely open and the pressure read ; after that the left pleura was laid open, and the pressure read again.

The observations were made upon the dead body.

1. Where the lungs are healthy the opening of one side of the chest goes a long way to satisfy the elasticity of both lungs, as the following figures show :—

Pressure on opening the right side, .	29	49	39	32
,, ,, left ,, .	11	5	9	7
Total pressure (right + left), .	40	54	48	39

This is very interesting, as it explains what we often observe in pneumothorax, viz., that the symptoms are the more severe the more healthy the patient has hitherto been ; for if we take the total elasticity in the adult man, according to these experiments, to be on the average 50,

the opening of the right side diminishes this elasticity by 40, leaving only 10 for the other; and therefore the respiratory capacity of such an individual would be reduced by the opening of one side to the extent of four-fifths—a difference which is not met with to the same amount where the lung is diseased.

2. Where the pleura is adherent the lungs are held back, and the recoil is thus prevented more or less, and sometimes entirely.

If the pleura be universally adherent, the contractility of that lung (or of both, if both be affected in the same way) will be entirely abolished; for even if the elasticity be retained (and it seems to be retained sometimes), it will be prevented from acting. The degree to which the elasticity of the lung will be impaired depends upon the extent of the adhesions, and upon the secondary changes which they have produced in the lung tissue itself.

Where the pleura is partially adherent, the opening of the remaining portion of the pleura on the one side does not produce the same marked effect upon the total elasticity of the lung as in the case of healthy persons.

If one pleural cavity be completely obliterated, the adhesions being universal, and the side be greatly contracted, the elasticity of the lung on the opposite side may be greatly reduced; but, on the other hand, if the lung has undergone compensatory development, the elasticity may not only be not reduced, but may be actually increased. This is a very interesting and important observation, because it shows that the condition which is often described as "compensatory emphysema" is really a compensatory hypertrophy, as indeed there are the best clinical grounds for believing.

Thus, in a case of the kind, on the right side, where the pleura was completely adherent, the elasticity of that lung was reduced to 12; whilst on the opposite side (the left), where the pleura was free, although there was some slight chronic tubercular change in the apex, the elasticity of the lung was 57.

The contractility of the lung is in all probability not purely elastic, that is to say, it does not depend solely upon the elastic tissues in the lung, but probably also upon the muscular tissue with which the lung is so richly provided. Indeed, we may in this respect compare the lungs with the arteries and speak of "pulmonary tone" as we do of "arterial tone," and connect the tone in the lungs, as we do in the arteries, with the condition of the muscles, and possibly of the nerves.

If there be such a thing, therefore, as pulmonary tone, we might expect it to vary, as the tone of the vessels does, with the conditions of general health or of local disease, and there are some among Perl's observations which go far to prove this contention. Thus, without any local disease, the elasticity of the lungs was found greatly reduced in several cases of typhoid, in a case of severe hæmorrhage after ovariotomy, and in cases of delirium tremens, erysipelas, and phosphorus poisoning.

The condition would then be brought into close clinical association with that of acute tympanites, and, like it, would be a neuro-paralytic phenomenon, and of grave significance. For my own part, I believe there are good clinical grounds for believing that there is a neuro-paralytic condition of the lungs, strictly analogous with that condition of the abdomen called tympanites.

The two conditions might, of course, theoretically be associated, but when the abdomen is distended from any cause, whether it be from gas, effusion, or tumour, the displacement of the diaphragm upwards is so great that the tympanitic percussion obtained over the lungs may be due to mechanical rather than physiological or pathological causes.

Where the lungs are diseased, the other non-diseased parts may yield tympanitic resonance on percussion. This is a condition with which we have been long familiar under the name of Skodaic resonance. There are several conditions under which this phenomenon appears. The commonest and easiest to explain is that in which it is associated with an effusion into the pleura. In this case, as the fluid forms, the lung collapses and floats on its surface, and over this collapsed lung tympanitic percussion note is obtained. Its occurrence has been rightly referred to the relaxation of the lung tissue, and properly compared with the percussion note which is obtained over the lung removed from the body.

Again, conditions of complementary emphysema, where one part of the lung is collapsed and the other proportionately distended, may give rise to similar hyper-resonance.

But besides these there is, I believe, a third condition, and this requires a different explanation.

Nothing is much commoner with pneumonia than to find the parts of the lung above or in front of the affected portion yielding a highly tympanitic note, yet the pneumonic portions of the lung are certainly not collapsed or smaller than they should be, nor are they much larger; so neither of the explanations given is adequate, for where the hyper-resonance is obtained the lung is not collapsed on the one hand nor over-distended on the other. This condition, it appears to me, can only be explained on the assumption of a loss of lung-tonus, of neuro-paralytic origin, dependent on nutritive disturbance. This view is supported by some of Perl's

observations; for example, there are several cases of pneumonia and some of embolism and gangrene, and in all of them the elasticity of the lung was very greatly reduced.

Acute miliary tuberculosis provides, perhaps, a still more striking instance of this condition, for the hyper-resonant or tympanitic note on percussion may be almost the only physical sign of disease yielded by the lung, and there can be little risk of error in assuming that the nutrition of the lung is profoundly affected in this disease.

In acute bronchitis, again, both in the child and in the adult, this nutritive disturbance in the lung probably explains in great part the tympanitic percussion obtained; but proof is more difficult in these cases because of the other conditions which may take their share in producing it, viz., the collapse of some parts of the lungs and the complementary emphysema of others.

In estimating the intra-pleural tension and the elasticity of the lung, the condition of the abdomen cannot be disregarded. During life there is the respiratory action of the abdominal muscles to reckon with, and after death we have to consider the effects of rigor mortis in them.

It is possible that the elasticity of the lung diminishes after death, but there are no direct observations which prove this, though it may be that this is the explanation of the difference as between Perl and Donders in their estimate of the average value of the elasticity of the lung.

Under pathological conditions the two layers of the pleura may be separated either by air or by fluid, and these present each of them its own peculiarities; thus fluid has weight, but it is practically incompressible; while air is compressible, but its weight may be disregarded. With fluid, therefore, the height of the column of fluid above the point of the trocar would affect the manometer reading, while with air the position of the puncture is indifferent.

With air the difficulties of the problem are in many respects less, and it will be well to begin with that condition first.

1. INTRA-PLEURAL PRESSURE IN PNEUMOTHORAX.—The air may have gained access to the pleura either from without through the chest walls, or from within from the lung or some other air-containing viscus.

I. *Where there is an opening through the chest walls.*—(*a*) A punctured wound, such as is made by a stab with a penknife, where the lung is injured as well as the external walls, we need not consider, for in this case, though the lung is wounded, air does not make its way in most cases into the pleura, but crosses the pleura and reaches the subcutaneous tissue. The explanation of this is very difficult, but need not be considered here.

(*b*) Where the opening through the chest walls is a large one, say at least as large as the section of the trachea, the air will pass freely in and out during respiration without let or hindrance as easily as it does through the trachea. In this case the pressure on the two sides of the lung, viz., that in the trachea and that in the pleura, will be equal always throughout all phases of respiration. Then the elasticity of the lung is free to come into play and the lung collapses.

No doubt, as Donders said, the elasticity of the lung itself is able in time to produce complete collapse, but that this is not the result during life, daily observation demonstrates in cases in which the pleura is laid open by operation. In many of these cases where the opening is free, the lung, instead of being found completely collapsed, as might be expected, is on the contrary found distended to a certain point, and reaching, it may be, within an inch or so of the walls of the thorax; or if it has been collapsed, as for instance by an empyema, as soon as this has been evacuated the lung often rapidly expands so as to occupy the position just indicated.

Although it is true, when the respiratory movements are absent, as they are in the dead body, that the atmospheric pressure being equal on the two sides, the elasticity of the lung will lead to its complete collapse, this is not true while the respiratory movements are acting, for the air does experience some obstruction in the air-tubes, both on entrance and on exit; thus the pressure of the air in the air-tubes during inspiration is about half a millimetre of mercury ($\frac{1}{4}$ inch of water) below that of the atmosphere; while on expiration it is as much as 2 to 3 millimetres of mercury (1 to $1\frac{1}{2}$ inches of water) above it, and these differences are still greater if the respirations are violent and forced.

Under these circumstances it is clear that the lungs would only collapse until the pressure within them was equal to 2 or 3 millimetres of mercury, and that they would then cease to contract any more. If the lungs can be completely expanded under a pressure of 6 millimetres of mercury, they could certainly be not less than half expanded at any rate under a pressure of 3 millimetres. What the exact relative volume of the lung might be actually under a pressure of 3 millimetres of mercury there are no observations to show.

When the expirations are forced, as in coughing, straining, or vomiting, the expiratory pressure becomes much higher than 3 millimetres of mercury, and the lung may then, if the external opening be free, be forced out again into actual contact with the chest walls, or even made to protrude through the opening in the side.

Actual protrusion of the lung during coughing in these circumstances, however, rarely occurs, but the movements of the lung may be seen to be very free.

(*c*) Where the external opening is not large enough to allow the air to pass freely out during expiration, the excess of air which cannot escape is compressed, and the pressure rises above that of the atmosphere ; the lung is in consequence squeezed, and is more and more compressed until it becomes completely collapsed. The condition is then much the same as that in which the air has gained access to the pleura from the lung.

II. *Where the opening into the pleura is from within.*—Theoretically there might be a condition in which the opening into the lung was large enough to permit the air to pass freely in and out on respiration without obstruction. This, however, as a matter of fact, hardly ever occurs. Air practically always enters the pleural cavity more easily on inspiration than it finds exit from it during expiration.

The result is, as in the former case, that the lung becomes compressed during expiration, and the more rapidly so, the greater the difference between the pressures of inspiration and expiration.

The division of pneumothorax into open, closed, and valvular thus becomes of little value from the present point of view, for all pneumothorax is at first more or less valvular, *i.e.*, the air enters more easily during inspiration than it finds exit during expiration, and when the lung is completely collapsed, the pneumothorax practically becomes closed, whether the aperture be sealed permanently or not.

In pneumothorax the pressure conditions during inspiration and expiration require to be considered separately.

1. *The inspiratory pressure.*—When pneumothorax has occurred, air will continue to enter on inspiration until the pressure within the pleura is equal to that of the atmosphere ; as soon as this point is reached, no more air can enter, consequently with a simple pneumothorax the inspiratory pressure cannot exceed that of the atmosphere, *i.e.*, the reading on the manometer will be zero.

This is subject to one condition. In the early stages of pneumothorax the respirations are excessive, and air will enter the pleura until the pleural pressure is equal to that of the atmosphere on deep inspiration. When after a time the urgent dyspnœa has passed off, so that inspiration is not so deep, the pressure may be above that of the atmosphere to the extent of the difference between deep inspiration and ordinary inspiration ; probably to a great extent this relieves itself in time by the absorption of a sufficient amount of the air to remove the difference.

If the inspiratory pressure be permanently above that of the atmosphere, that is to say, is positive, there is required some other factor to cause the rise, and this, as a matter of fact, we find to be the development of effusion, so that, speaking generally, a positive pressure in pneumothorax during inspiration indicates the presence of fluid.

2. *The expiratory pressure.*—Except in the theoretical and rare conditions in which there is no obstruction to the exit of air from the pleural cavity during expiration, the expiratory pressure in pneumothorax will always be positive in the early stages.

In these cases the heart and mediastinum are found displaced to their maximum and the lung is completely collapsed.

The collapse of the lung, though capable of being brought about in time by its own elasticity, is always in chief part due to the compression exercised by the rise of expiratory pressure.

In the same way the displacement of the mediastinum, though capable of being brought about by the elastic traction of the opposite lung alone, is assisted by the raised expiratory pressure and caused to develop more rapidly or to be more extreme.

The respiratory oscillation.—The respiratory oscillation is the difference between the pressure on inspiration and the pressure on expiration, and this presents also some interesting points for consideration.

If the movements were violent the respiratory oscillations would, it might be thought, be considerable ; but as a rule this is not so, for on the affected side the chest is in the position of maximum inspiratory distension, so that no further expansion on inspiration is possible ; while on the opposite side, owing to the displacement of the heart and mediastinum, the elasticity of the sound lung is reduced, so that the respiratory excursion is diminished, the result being that even with considerable dyspnœa the respiratory oscillation is not only not raised above the normal, but may be considerably below it. It is, indeed, upon the defective respiratory excursion of the two sides that much of the dyspnœa depends.

Yet there are cases in which extensive respiratory oscillations are seen, and it is important to consider the interpretation which may be given of them.

The number of cases of pneumothorax in which I have estimated the pressure in the pleura is 11. In several of these paracentesis was performed more than once and the pressure determined, so that I have returns of the pressure in 20 paracenteses. In most of them I am able to record the inspiratory pressure, the expiratory pressure, and the difference between them, *i.e.*, the respiratory oscillation.

In two cases the inspiratory pressure was that of the atmosphere, *i.e.*, the manometer reading stood at zero at the time of the operation. In both these cases fluid was present as well as air, and from this it is evident that, as the fluid formed, the air must have been absorbed, since the opening in the lung in both cases was closed.

In another case the inspiratory pressure, after having been in the first two paracenteses positive, in the last two fell to zero, and in this case the change in pressure was due to the opening in the lung having become patent and being of considerable size.

In all the other cases the inspiratory pressure was positive, and fluid was present as well as air—sometimes pus, sometimes serum. Although, as I have already stated, the inspiratory pressure in the early stages of pneumothorax may be positive to some extent, it can never be high ; and practically we may conclude that when the inspiratory pressure is much raised, fluid is present at well as air in the pleura. This was at any rate the fact in the instances recorded.

The range of pressures in the different cases is considerable—thus the inspiratory pressure varied from 0 to 9 inches, the actual pressures being $0\frac{1}{2}$, 1, $1\frac{1}{2}$, $2\frac{1}{2}$, 4, $4\frac{1}{2}$, 5, $6\frac{1}{2}$, $6\frac{3}{4}$, $6\frac{7}{8}$, 7, $8\frac{1}{4}$, 9 ; the expiratory pressure varied from 0 to $13\frac{1}{4}$, the respiratory oscillation from 0 to $6\frac{5}{8}$.

The amount of fluid removed is no indication of the amount of fluid present, for the effusion lies in the diaphragm as it were in a saucer, and unless the point of the trocar be carefully manipulated, so as to be beneath the level of the fluid, air will escape, the pressure fall, and no fluid be removed unless suction be used.

Suction, and especially the use of the aspirator, is in all cases of pneumothorax risky ; for where the hole is patent, it is useless except for the purposes of removing fluid, and then the trocar must be carefully manipulated ; and if the opening be closed, suction is very likely to tear it open afresh.

The aspirator, therefore, is a dangerous instrument in pneumothorax, and should be rarely employed.

The ordinary syphonage apparatus, if properly manipulated, is all that is required and is devoid of risk.

It is not to be wondered at that the inspiratory pressure rises, when fluid forms, in pneumothorax, but it is surprising that the pressure is not much higher than it is. The highest pressure that I have to record is 7 inches of water ; and a pressure as high, or even higher, is met with in simple serous effusions.

It follows, therefore, that when fluid forms in pneumothorax a large amount of the air present must be absorbed as the fluid forms.

That the absorption of air from the pleural cavity is very rapid we know from experiments on animals, and also from observations made in cases of accident and operation in man.

There are cases of pneumothorax even in man, in which, in spite of the presence of fluid, the inspiratory pressure is not above that of the atmosphere, as in the two cases mentioned.

There are two conditions under which the pressure may rise rapidly in an ordinary pneumothorax :

(*a*) Where the effusion is poured out rapidly ; the fluid must then be serous in character, so that time enough has not been given for the air to be sufficiently absorbed.

(*b*) Where the lung or pleura is so far affected that the absorption of air does not take place as readily as it should. This no doubt is the fact in most cases of pneumothorax ; and I think we shall not be going too far if, in cases of pneumothorax of long duration, we accept the existence of a high intra-pleural pressure as an indication of somewhat extensive disease of both lung and pleura.

In two cases the pressure was determined at the end of the paracentesis, as well as at the commencement of it. In the one case no suction was employed and the pressure fell to that of the atmosphere, the manometer registering zero. In another case some suction was used, and at the end of the operation the pressure registered – 9 inches of water. In another case the pressure, which on the first two paracenteses had been positive, in the third and fourth proved to be zero. The aspirator was then used, nothing but air was removed, and as this had no effect upon the pressure it was evident that the air passed freely into the pleura from the lung ; in other words, that the opening was patent. This proved to be the case on the autopsy.

Further comment will be most usefully made in connection with the description of each case.

No. of case.	Sex.	Age.	Side.	I. Pr.	E. Pr.	R. O.	Amount of fluid removed.	Remarks.
							oz.	
1	M.	31	R.	0	0	0	80, turbid serum	11 weeks' duration; ultimately recovered.
2	M.	31	L.	0	$1\frac{1}{2}$	$1\frac{1}{2}$	11, pus	3 weeks' duration; marked phthisis.
				0	8	8	10	Great dyspnœa; 2nd paracentesis 7 weeks after the first; ultimate incision; necrosis of 6 inches of rib; death from exhaustion.
3	M.	30	L.	$1\frac{1}{2}$	$2\frac{1}{2}$	1	Pus	3 weeks' duration; at end of operation; Pr. = − 9; ultimately side discharged; incision; death.
4	M.	24	L.	$8\frac{1}{4}$	—	—	34, pus	3 weeks' duration.
				—	—	—	41, pus	14 days later.
				7	$8\frac{1}{2}$	$1\frac{1}{2}$	Much	3 days later; free opening formed in lung; incision subsequently; death.
5	M.	22	R.	$6\frac{7}{8}$	$13\frac{1}{4}$	$6\frac{5}{8}$	41, sero-pus	3 months' duration.
				$6\frac{1}{2}$	8	$1\frac{1}{2}$	22, sero-pus	3 weeks later; ultimate recovery.
6	M.	21	R.	1	5	4	28, sero-pus	5 weeks' duration.
				$\frac{1}{2}$	$2\frac{1}{4}$	$1\frac{3}{4}$	36	17 days later; subsequent incision; death from hæmoptysis. *P.M.* Ruptured pulmonary aneurysm.
7	M.	24	L.	4	5	1	22, serum	3 days' duration; recovery.
8	M.	35	L.	$6\frac{3}{4}$	7	$\frac{1}{4}$	6, pus	Many months' duration. At end of operation I. Pr. = 0, R.O. = 2. Was pointing at time; therefore pointing pyopneumothorax may have low or even zero pressure. Interesting case to compare with empyema.
9	F.	23	L.	$2\frac{1}{2}$	9	$6\frac{1}{2}$	Several, pus	3 days' duration. Much dyspnœa at time of operation. At end of operation R.O. = 1, dyspnœa passed off.
				$4\frac{1}{2}$	5	$\frac{1}{2}$	—	3 days later, owing to little fluid being obtained, suction employed up to − 36 inches of water, but only 6 oz. more fluid obtained. ∴ Lung bound down. Incision. Death from shock immediately on incision. No *p.m.*
10	M.	46	—	9	9	0	74	10 days' duration.
				1	$4\frac{1}{2}$	$3\frac{1}{4}$	Serum	16 days later, on deep Inspir. Pr. = − 1; on cough Exp. Pr. = 7. Ultimately puncture suppurated, though fluid was serous. Patient lived 12 months, and died of phthisis.
11	M.	22	L.	5	$8\frac{1}{2}$	$3\frac{1}{2}$	36, serum	A few days.
				1	6	5	A few	9 days later.
				0	0	0	—	Opening had become patent.
				0	$\frac{1}{2}$	$\frac{1}{2}$	40, pus	On death large patent opening found in lung.

2. **INTRA-PLEURAL PRESSURE IN SEROUS EFFUSION.**—In health the pleural cavity contains no fluid, and we often speak of it as dry; yet this is inaccurate, for there is in all probability a constant circulation of fluid into the pleura and out of it, the fluid being effused by the blood-vessels and carried away by the lymphatics. The mechanism by which this is performed has been described as the *lymphatic pump*. It consists of the lymphatic vessels with their stomata and valves, and is worked by the respiratory movements. The course of the circulation in the lung is from the pleural surface (the periphery, that is to say), towards the root of the lung, as has been determined by experiment, and there is a similar circulation from the pleural surface through the diaphragm and through the chest walls.

There are two ways, therefore, in which fluid may accumulate in the pleura; either it may be poured out into the pleura in larger quantities than the pump can remove, or the amount of fluid poured out may not be above the normal, but the pump be defective.

In the case of pleural inflammation both these processes probably come into play; the amount of transudation is considerable, and the stomata and smaller lymphatics are often plugged by a deposit of fibrin. Thus in inflammatory cases the fluid may accumulate with very great rapidity and soon reach a large amount.

In a case of dropsy of the pleura consequent, for instance, on heart disease, the explanation is probably also mechanical. Exudation under these conditions takes place from the blood-vessels into the lymphatics of the lung, which become water-logged or choked, and thus unable to carry off the fluid from the pleural cavity, so that it accumulates in it.

With dropsy, however, the development of fluid is much slower and the amount as a rule much less.

When fluid collects in the pleura it falls by its weight to the lowest part, and although the tension in the whole pleural cavity will be diminished in proportion to the amount of fluid present, still the effect upon the different parts of the lung will be different; thus the lowest parts will suffer most and become collapsed, as we know they do, while the upper parts of the lung remain distended; but the tension in the upper part of the pleural cavity is lower than it otherwise would be, as is shown by Calvert's observations (*St. Barthol. Hosp. Rep.*, 1892, p. 131).

This diminished tone in the lung or tension in the pleura explains the hyper-resonant note which is obtained in those parts of the lung which are floating upon the fluid.

In determining the intra-pleural pressure in cases of fluid effusion, something will depend upon the seat of puncture, as Calvert also has shown, for if the mouth of the trocar be 1, 2, or 3 inches respectively below the level of the fluid, there will be the pressure of a column of fluid of this height to allow for.

If, for example, the intra-pleural tension be equivalent to 3 inches of water, and the amount of fluid exuded into the pleura be sufficient to reduce this 3 inches negative pressure to 2 inches negative pressure, it follows that if the mouth of the trocar be 2 inches below the level of the fluid, a positive pressure of 2 inches will have to be added to the negative pressure in the rest of the pleura, which will reduce the pressure-reading to zero; or if the height of the fluid be 3 inches instead of 2, will convert the pressure at the point of puncture to a positive pressure of 1 inch.

In most of my cases I have taken as far as possible the same relative position in the chest for puncture, viz., the middle of the axilla, as the patient is lying upon the back; the results are therefore more or less comparable with one another; but although I have endeavoured to make some allowance for these considerations, the readings in pleural effusions have not anything like the same value as those in pneumothorax.

I have determined the pressure in 27 cases of serous effusion, some of which were tapped more than once, so that I have 31 pressure-records to found these observations upon.

In the table on the following page I have arranged the cases in order according to the size of the effusion.

A glance at this table will show that the pressures vary greatly and irregularly, and that there is no definite relation between the size of the effusion and the amount of pressure; for there are among the cases instances of large effusions with low or even negative pressures, and of small effusions with high pressures.

Thus a **large** effusion was found:

		Amount.			Pressure.
	With *negative* pressure in—				
Case 3	... ...	112	...	...	−1
	A few days later	70	...	...	−1
	With *low* pressure—				
,, 26	... ...	large	...	...	1
,, 1	... ...	139	...	...	$2\frac{1}{2}$

No. of case.	Sex.	Age.	Side.	Amount of fluid removed.	Pressure.	Resp. osc.	Remarks.
				oz.			
1	M.	46	R.	139	$2\frac{1}{2}$	0	At end of operation Pr. = $-9\frac{1}{2}$; resp. osc. = $\frac{1}{2}$; spontaneous coagulation in fluid.
2	M.	41	—	130	18	$\frac{4}{10}$	
3	M.	32	L.	112	-1	3	
				After 6 days 70	-1	1	
				,, ,, 72	$+3\frac{1}{2}$	1	
4	M.	39	L.	106	8	$1\frac{1}{4}$	After paracentesis fluid rapidly disappeared; Pr. after first paracentesis = -1.
				19	5	$1\frac{1}{2}$	
5	M.	35	L.	98	4	1	
6	M.	39	—	85	6	$\frac{1}{4}$	Fluid apparently formed in 5 days.
7	M.	51	R.	77	5	2	Blood-stained fluid.
8	Cf.	Case	3	72	$3\frac{1}{2}$	1	
9	M.	42	L.	72	$1\frac{1}{2}$	1	
10	Cf.	Case	3	70	-1	1	
11	M.	22	L.	68	$8\frac{1}{2}$	—	
12	M.	33	R.	62	0	$1\frac{1}{2}$	On second paracentesis Pr. = 0; resp. osc. = 8 (from $+4$ to -4); a few ounces of pus.
13	M.	54	L.	60	0	0	Died shortly after of acute phthisis.
14	M.	63	R.	55	4	2	
15	F.	25	R.	52	$8\frac{1}{2}$	1	Sp. gr. of fluid 1040; spontaneous coagulation.
16	M.	34	R.	50	$2\frac{1}{4}$	—	
17	F.	29	L.	47	2	$\frac{3}{4}$	
18	M.	83	L.	45	11	$1\frac{1}{4}$	
19	F.	46	L.	46	11	—	Fluid blood-stained.
20	M.	23	L.	40	0	$\frac{3}{4}$	100 ounces removed previously, but pressure not taken.
				20	3	$\frac{3}{4}$	
21	F.	—	—	40	$1\frac{1}{4}$	$\frac{3}{4}$	
22	—	—	—	34	0	2	
23	F.	53	L.	30	$\frac{1}{2}$	$\frac{1}{2}$	Sudden death. Pleurisy secondary to sarcoma of vertebræ.
24	Cf.	Case	19	20	3	$\frac{3}{4}$	
25	Cf.	Case	4	19	5	$1\frac{1}{2}$	
				Amount not specified.			
26	F.	34	—	Very large	1	$\frac{3}{4}$	
27	M.	26	L.	Large	8	—	
28	M.	—	L.	Moderate	0	2	
29	M.	62	L.	Large	$11\frac{1}{2}$	—	At end of operation Pr. = $+3$.
30	M.	46	L.	Small	0	—	Cast expectoration subsequently from trachea. Death.
31	F.	19	—	Moderate	-4	$\frac{1}{2}$	

		Amount.		Pressure.
	With *high* pressure—			
Case	2	130	... ...	18
,,	4	106	... ...	8
,,	5	98	... ...	4
,,	6	85	... ...	6
,,	27	large	... ...	8
,,	29	large	... ...	$11\frac{1}{2}$

In the same way with **moderate** effusions the pressure may be negative, zero, moderate, or high.

	With *negative* pressure—			
Case	10	70	... ...	−1
	With *zero* pressure—			
,,	12	62	... ...	0
,,	13	60	... ...	0
,,	28	moderate	... ...	0
	With *high* pressure—			
,,	7	77	... ...	5
,,	11	68	... ...	$8\frac{1}{2}$
,,	15	52	... ...	$8\frac{1}{2}$
,,	14	55	... ...	4

Small effusions in the same way :

	With *zero* pressure—			
Case	20	40	... ...	0
,,	22	34	... ...	0
	With *low* pressure—			
,,	23	30	... ...	$\frac{1}{2}$
,,	21	40	... ...	$1\frac{1}{4}$
	With *high* pressure—			
,,	18	45	... ...	11
,,	19	46	... ...	11
,,	24	20	... ...	3
,,	25	19	... ...	5

If we look at these cases again from the point of view of **pressure,** we find :

A **negative** pressure—

	With *large* effusion in—			
Case	3	112	... ...	−1
	With *moderate* effusions in—			
,,	10	70	... ...	−1
,,	31	moderate	... ...	−4

A **zero** pressure—

	With *moderate* effusions in—			
Case	12	62	... ...	0
,,	13	60	... ...	0
,,	28	moderate	... ...	0
,,	30	small	... ...	0
	With *small* effusions in—			
,,	20	40	... ...	0
,,	22	34	... ...	0

Very high pressures—

	With *large* effusions in—			
Case	2	130	... ...	18
,,	29	large	... ...	$11\frac{1}{2}$
,,	4	106	... ...	8
	With *moderate* effusions in—			
,,	11	68	... ...	$8\frac{1}{2}$
,,	15	52	... ...	$8\frac{1}{2}$
	With *small* effusions in—			
,,	18	45	... ...	11
,,	19	46	... ...	11

It might be thought that this irregularity could be explained by the different stages in which the inflammatory effusions were at the time of investigation; for instance, that where the inflammation was active, the pressure might be high; where the inflammation was subsiding, the pressure might be low; and that when the fluid was being absorbed, the pressure might even become negative.

Case 6 might, perhaps, bear this interpretation, for the fluid in this case was accumulating with very great rapidity, and within four or five days from the very commencement of the illness, 85 ounces of fluid were removed from the pleura; the pressure in that case was + 6; but this theory is not supported by many other cases, and I am afraid the whole matter is by no means so simple as this would make it.

Two cases present a special interest because the paracentesis was repeated, and the pressure determined on subsequent occasions. Case 3 is an instance of the kind. On the first paracentesis 112 ounces were removed, and the pressure was −1. Six days later 70 ounces more were removed, and the pressure was −1 again. Six days later still, 72 ounces were removed, and then the pressure was $+3\frac{1}{2}$.

Case 19 is another instance of the kind. On the first paracentesis 100 ounces were removed, and the pressure was not determined. On the second, 40 ounces were removed, and the pressure was zero. On the third, 20 ounces were removed, and the pressure was +3.

It is tempting to suppose that by the third paracentesis in each of these cases the fluid had become encapsulated, the pleura being adherent elsewhere, and that this might account for the curiously sudden rise in pressure which occurred in both cases.

In three cases the pressure was determined at the end of the operation as well as at the commencement of it. In Case 1, where 139 ounces were removed, the pressure at first was $2\frac{1}{2}$, while at the end of the operation it was $-9\frac{1}{2}$. In Case 4 the pressure at the commencement of the operation was 8, and at the end of the operation −1; and in Case 29, where the pressure was $11\frac{1}{2}$ at the commencement, it still remained +3 at the end.

The Respiratory Oscillation.—The respiratory oscillation is, as alreay stated, in health about 1 to $1\frac{1}{2}$ inches of water, and in 15 of the cases recorded the respiratory oscillation was of about this amount.

It was reduced to zero, that is, there was no respiratory oscillation at all in 3 cases: Case 1 with 139 ounces of fluid; Case 12 with 62 ounces of fluid; and Case 13 with 60 ounces of fluid.

It was also below 1 inch in 9 cases. On the other hand, it was increased only in 1, viz., Case 3, in which with 112 ounces of fluid and a negative pressure, −1, there was a respiratory oscillation of 3 inches.

The respiratory oscillation, therefore, is generally reduced in serous effusions, and may be completely absent.

Inasmuch as the action of the lymphatic pump depends upon the respiratory movements, of which the respiratory oscillation is the index, it is evident that where the respiratory oscillation is absent the mechanism for the removal of fluid must be defective.

Where, with a large effusion at the time of introducing the trocar, the respiratory oscillations are seen to be absent, they frequently return after the fluid has been removed, and may even be above the normal. Even the partial removal of the fluid, *i.e.*, the removal of a part of it and not the whole, may cause the respiratory oscillations to return. This explains what is frequently observed and is difficult otherwise to understand, viz., that the removal of a small quantity of fluid not infrequently leads to the rapid spontaneous disappearance of the rest; the interpretation is that the lymphatic pump has been started to work again.

Other points which these tables show are that pressures have no relation to age, for the cases range from the young to the old; nor to sex; nor to the side affected; nor is the pressure any help in prognosis, for nearly all the cases in this list recovered with the exception of three, one of which died of rapidly advancing phthisis, the second of cancer, and the third with sarcoma of the vertebræ.

These observations have a bearing also upon the mechanism of the displacement of the heart and mediastinum in pleuritic effusion, for they show that displacement does not necessarily depend in any way upon pressure, for the maximum displacement may be found with a zero pressure or even with a negative pressure on the affected side.

We may conclude that the intra-pleural pressure in serous effusions is not as simple as it might *a priori* be thought to be; that it depends upon many factors, which are difficult to allow for, and that the whole subject requires further investigation.

3. INTRA-PLEURAL PRESSURE IN EMPYEMA.—I have ten observations of the pressure in empyema. They show that the question of intra-pleural pressure in empyema is much simpler than in the case of serous effusion, and completely in accord with what we should expect from our knowledge of suppuration in other parts.

We know that in other places the formation of pus is attended with considerable tension—in other words, that it goes on under considerable pressure. It is only in the very chronic, so-called cold abscesses that the tension is low; but even then the pressure is probably considerably above that of the atmosphere.

No. of case.	Sex.	Age.	Amount removed.	Pressure.	Resp. osc.	Remarks.
			oz.			
1	M.	34	91	16	0	
			56	10	0	Subsequently expectorated pus. Developed pneumothorax and died with phthisis.
2	M.	5	6	4	0	After whooping-cough.
3	F.	19	2	3	$\frac{1}{2}$	
4	M.	28	$14\frac{1}{2}$	$5\frac{1}{2}$	–	
5	M.	29	20	3	1	
6	M.	$6\frac{1}{2}$	40	8	$\frac{1}{2}$	
7	M.	26	15	8	0	On subsequent paracentesis 15 ounces removed twice; incision and recovery.
8	F.	13	15	5	$\frac{3}{4}$	On subsequent paracentesis 6 ounces and 1 ounce removed; cure by paracentesis only.
9	M.	35	6	7	$\frac{1}{4}$	

It will be seen that among my observations in all cases the pressure is considerably increased. The lowest of the series is 3, and the highest 16.

The highest pressure is found, as it happens, with the largest effusion, for in this case 91 ounces were removed with a pressure of 16, and on a subsequent paracentesis 56 ounces were removed with a pressure of 10.

There is, however, no necessary relation between the size of the effusion and the height of the pressure; for if small empyemata are localised, as they frequently are, the pressure may be very high, though the amount of pus contained in them be very small.

There is one interesting case, which bears upon this, among the observations in serous effusion, where a serous effusion of some size was tapped, but the pressure not raised.

On the third paracentesis the pressure was found to be +3, but the effusion then was no longer serous but purulent, the conclusion being that the serous effusion had been followed by a small local empyema. This was incised and the patient recovered.

The **Respiratory Oscillation** in empyema also is interesting; in all cases it was reduced, and in many of them absolutely disappeared. In four cases it was zero, in one $\frac{1}{4}$, in two $\frac{1}{2}$, and only in two instances did it approach 1.

The two points, therefore, in reference to the pressures in empyema are, first, that it is always positive, and sometimes considerably raised; and secondly, that the respiratory oscillation is practically absent.

58. CLASSIFICATION OF THE AFFECTIONS OF THE PLEURA.

The connective tissue of the pleura is continuous with the connective tissue of the adjacent parts, just as the lymphatics are. As a consequence of this the pleura is very liable to become involved in any disease which exists in its neighbourhood, so that a very large number of the affections of the pleura are secondary, and not primitive, *i.e.*, not of primary origin in the pleura itself.

The pleura may be compared with a joint, for its most important function is to render the movements of the lung free and easy during the expansion and contraction of the chest in respiration; and as in a joint, so in the pleura, there are three very important groups of affections. First those in which the joint is obliterated by adhesions; secondly, those in which the serous sac contains fluid; and thirdly, those in which the serous membrane itself is infiltrated and thickened.

"Pleurisy" is the general term given to all affections of the pleura, irrespective of their nature and cause, and "pleuritic" is the adjective used in the same general sense; thus we speak of "pleurisy with adhesion," or "pleuritic adhesion"; of "pleurisy with effusion," or "pleuritic effusion"; and of "pleurisy with thickening," or "pleuritic thickening."

Many pleurisies are the result of inflammation arising either in the pleura itself, or in the adjacent tissues.

The term "pleuritis" is used to indicate pleurisies of inflammatory origin, and the terms "acute" or "chronic" are added according as the inflammation is of recent origin and attended with acute symptoms, or not. Most of the acute affections of the pleura are inflammatory in origin. Many of the chronic affections of the pleura, however, are either the result of inflammation long past or are not of inflammatory origin at all.

Of pleuritis acuta there are two main forms, according as effusion is present or not, namely, "pleuritis sicca," and "pleuritis exudativa."

What is called "pleuritis chronica," though sometimes an inflammation of chronic course, *i.e.*, slowly progressive and of long standing, is in most cases a permanent pathological condition left by past inflammation, and then cannot be distinguished from similar conditions of other than inflammatory origin; so that this group is better described by the term "chronic pleurisy."

Of chronic pleurisy, again, there are three varieties—first, that associated with thickening of the pleura—"indurative pleurisy"; secondly, that leading to adhesion—"symphysis pleuræ"; thirdly, that associated with effusion, for example, "hydrothorax."

Pleurisies may be classified also in other ways; first, according to the nature of the effusion present, and thus we speak of "serous," "sero-fibrinous," "hæmorrhagic" and "purulent" pleurisy, the latter being also called "empyema"; secondly, according to its cause, and so we speak of "tubercular," "traumatic," "rheumatic," "syphilitic," "metapneumonic" pleurisy and others; thirdly, according to its localisation, *i.e.*, whether the pleurisy be general or localised, and so we have the varieties "basic," "diaphragmatic," "interlobar," "loculated," "encysted," "areolar," etc.; lastly, there are one or two affections of the pleura which do not conveniently fit into any classification at all, such as "hydatid," "cancer" and "pneumothorax."

It is evident that these various classifications cut into one another; that some are etiological, some pathological or anatomical, and some clinical. In practice the classification that is most convenient and useful is that which is clinical, and which follows closely the steps we take in making our diagnosis of a case.

At the bedside, given a case of pleurisy, the diagnosis usually proceeds in the following way. We endeavour to ascertain—

1. What the pathological condition is, *i.e.*, whether there is effusion, adhesion, or thickening?

2. If there be effusion, what its nature is; and lastly
3. What is the cause?

Affections of the pleura, whatever their cause may be, result in four prime morbid changes in the pleura.

1. The surfaces lose their polish and become rough, so that they do not move smoothly upon each other. Of this the characteristic physical sign is friction, and the characteristic symptom pain.

2. The surfaces become adherent to one another, thus causing some contraction and defective movement in the region affected.

3. The pleura becomes thickened. Besides some impairment of resonance on percussion, this leads to diminution of the voice- and breath-sounds.

4. Fluid collects in the pleural sac.

Physical examination usually determines without difficulty which set of changes is present. Assuming that we have a case of pleurisy to deal with, if there be no dulness on percussion it is clear that the pleurisy must be dry, and this opinion will be confirmed by the presence of friction; if the region be impaired or dull on percussion, the case will be one of pleuritic thickening or pleuritic effusion; in either case, as a general rule, the vocal vibrations, vocal resonance, and breath sounds will be diminished or absent, and the more marked the diminution and the greater the dulness, the greater the probability of effusion. If a large quantity of fluid be present, besides the general signs of fluid there will be evidence of the displacement of organs.

The *nature of the effusion* cannot be determined with certainty by physical examination alone, though its probable nature may be guessed from the general symptoms. Certainty can be arrived at, in most cases, only after some of the fluid has been removed with the needle by an exploratory puncture.

The cause of the disease during life, in any given case, can only be determined by careful consideration of the general condition of the patient, the previous history of the case, and the course the disease runs.

CLINICAL CLASSIFICATION.

A. The first great clinical group is formed of **the acute inflammatory pleurisies,** *i.e.*, **pleuritis acuta,** of which there are three main forms.

1. **Pleuritis sicca.**
2. **Pleuritis exudativa sero-fibrinosa.**
3. **Pleuritis exudativa purulenta,** *i.e.*, **Empyema.**

B. Of the **pleurisies not obviously inflammatory** most are **chronic,** or at any rate are not associated with acute symptoms. These again fall into three groups.

1. Those **with effusion;** (dropsical, hæmorrhagic or chylous).

a. **Hydrothorax.**
b. **Hæmorrhagic pleurisy,** and **hæmothorax.**
c. **Chylous pleurisy (Chylothorax).**

2. Those with **obliteration of the cavity.**

Symphysis Pleuræ.

As most of these cases are regarded as the result of chronic or past inflammation, they are often described under the name of pleuritis chronica.

3. Those in which, with or without obliteration of the cavity, there is much **thickening of the pleura.**

Pleuritic Thickening.

C. Besides these there are some **special forms of pleurisy** which call for consideration because of their importance.

a. In respect of *cause*;

Tubercular,
Metapneumonic,
Rheumatic,
Syphilitic, etc.

b. In respect of *localisation*;

Diaphragmatic,
Apex,
Double, etc.

The series of the affections of the pleura will be completed by the addition of—

New Growths,
Syphilis,
Actinomycosis,
Hydatid,
Pneumothorax.

It is under these various headings that the diseases of the pleura will be dealt with.

59. THE ACUTE INFLAMMATORY PLEURISIES.

GENERAL PATHOLOGY.—There are three forms of acute inflammatory pleurisy—pleuritis sicca, pleuritis sero-fibrinosa, and empyema.

Whatever may be the difference in etiology, *i.e.*, in the cause of the disease, the anatomical changes produced in the pleura in these three forms are much the same. They differ *inter se* rather in degree than in kind.

They are of three kinds:

1. Lesions of the serous membrane itself; hyperæmia and infiltration of the tissue, with nutritive changes in the epithelial cells.
2. Plastic exudation upon the surface of the membrane.
3. Effusion into the cavity, which may be serous, sero-fibrinous, hæmorrhagic, or purulent; the different forms being in some degree a measure of the different intensity of the inflammation, and to some extent also suggestive of the cause.

1. Lesions of the serosa.

The surface has lost its transparency and polish; it looks dull and cloudy, and is roughened and granular instead of smooth.

The colour is pink, or redder than normal, and arborescent groups of distended vessels are often visible. There may also be extravasations of blood into the substance of the membrane, either small and punctate, or larger and ecchymotic.

The substance of the membrane itself is also slightly thickened.

Microscopical examination shows that the epithelial cells are swollen and desquamating, and that active proliferation of cells is taking place, especially round the stomata.

The whole tissue is swollen, œdematous, and infiltrated with cells, especially near the surface, where the active proliferation taking place produces the small rough granulations referred to.

These granulations consist of embryonic cells with red and white blood cells, and they often contain newly-formed capillaries.

The granulating surface is often described as "new membrane," to distinguish it from the "false membrane," which is merely plastic exudation upon the surface.

It is the new membrane which becomes organised, and leads to thickening or to adhesion, while the pseudo-membranes probably do not undergo organisation at all.

The stomata are often filled with plugs of fibrine, which may extend some distance into the lymphatics with which the stomata communicate. The deeper lymphatics also are stuffed with white cells, and this infiltration may extend some distance into the tissues beneath the inflamed pleura.

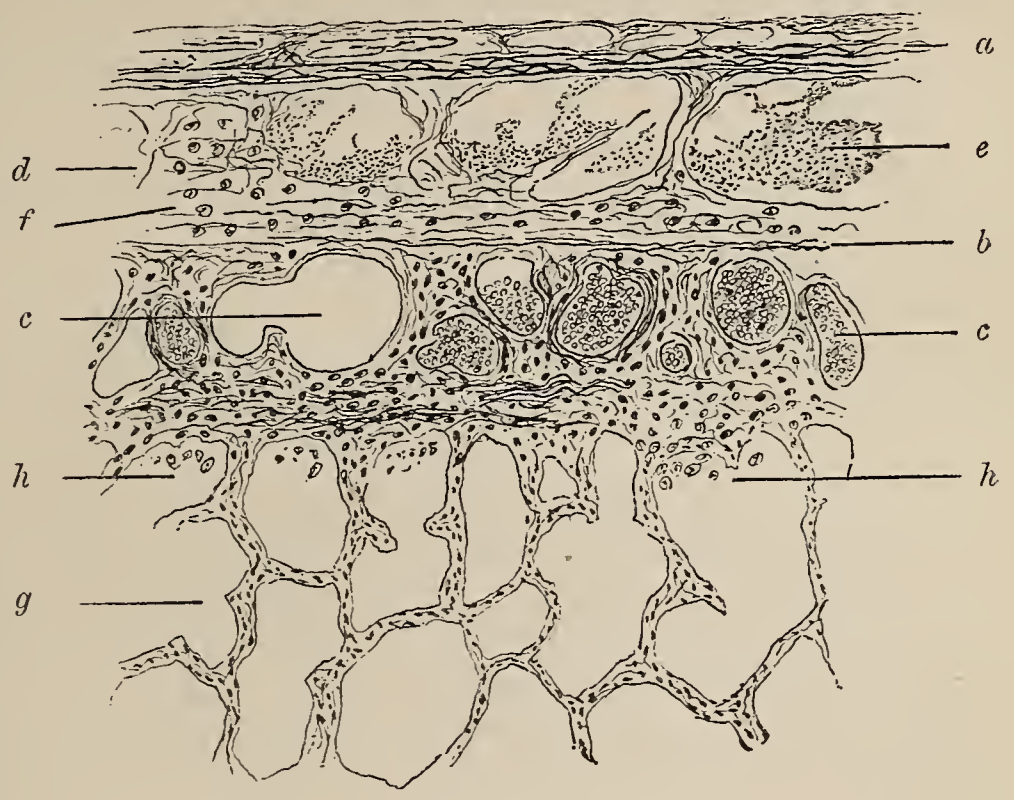


Fig. 144.

Section of an acutely inflamed pleura. *a*, parietal pleura; *b*, pulmonary pleura, much increased in thickness, infiltrated with cells, and containing numerous irregularly dilated blood-vessels (*c*); *d*, space between the parietal and pulmonary pleura, filled with exudation (*e*) in irregular spaces. Some of the meshwork is becoming organised into fibrous tissue (*f*); *g*, vesicles of the lung; those nearest to the pleura contain small cells (*h*), *i.e.*, are in a condition of early inflammation. Their walls are also infiltrated and thickened.

2. Plastic exudation.

This forms the false membrane lying on the surface of the pleura, or the shreds which float loose in the cavity when fluid is present. They are dull, opaque, grey, or, if thick, yellow in colour. Their consistency is usually gelatinous, like white of egg, but they may be firmer, like blood clots, though in any case easily torn. They are composed of layers of fibrine, enclosing in their meshes white and red blood cells and altered epithelial cells.

The exudation may form a smooth membrane of some thickness, even up to 1 cm., *i.e.*, two-fifths of an inch, and of considerable extent. The surface is more frequently reticulated rather than smooth, the result, no doubt, of the

movements of the pleural surfaces upon each other. When fluid is present, the exudation may take the form of long shreds, extending from one side of the pleura to the other, or of filaments attached at one side only, or portions may become detached and float free in the fluid, or sink as a sediment to the lowest part.

As already stated, these false membranes or plastic exudations do not become organised.

When there is no effusion, the changes just described are well named "plastic pleurisy" or "pleuritis sicca."

3. Effusion.

The effusions are of three kinds:—

(1) Serous or sero-fibrinous, *i.e.*, serum containing more or less of fibrine.
(2) Hæmorrhagic, *i.e.*, mixed with more or less of blood.
(3) Purulent.

Empyema differs in so many important respects from other forms of effusion, that it will be better to defer entirely what has to be said about its pathology until later.

The inflammatory forms of hæmorrhagic effusion again differ only from the sero-fibrinous in the larger proportion of blood which they contain, so that they may be regarded, as far as the pathology goes, as differing only in degree and not in kind.

Serous and sero-fibrinous effusion.—The fluid is usually quite clear and almost perfectly transparent, of a pale lemon or sherry colour, and without smell.

Sometimes it is a little turbid, like whey or water from a milky jug, owing to the admixture of a small number of white cells or pus cells. It may contain a few flakes of plastic exudation.

Sometimes it is rosy or pink, and occasionally it may be even dark or porter coloured. This is the result of the presence of blood. Some red blood cells are present in every serous or sero-fibrinous effusion, but they do not give a colour to the fluid till they exceed 4000 or 6000 in the cm.

The dark colour is due to a considerable admixture of blood, and to the fact that the blood has been present some time in the fluid, and has undergone the usual decomposition colour-changes.

The specific gravity varies from 1012 to 1022, but usually it approaches rather the lower limit. It is stated that the higher the specific gravity the more favourable is the prognosis.

Thus Lemoine says that it is favourable if the specific gravity be above 1019, and unfavourable if it be below 1015; this, however, is not my experience.

The effusion contains about 57 to 70 parts of solid substances in the 1000, *i.e.*, with 943 to 930 parts of water.

	Water.	Albumen.	Fibrine.	Salts.	Organic substances.	Total solids.
Simon,[1] .	947	31	1	19	12	63
Mehu,[2] .	943–930	10–15	rarely above 1	7–10	...	50–60

The inflammatory effusions, as compared with the dropsical, are of higher specific gravity, and are richer in proteids and in fibrine, and approach more nearly to the constitution of lymph than of serum.[3]

	Inflammatory.	Dropsical.
Specific gravity,	1021	1014
Total proteid per cent.,	4·59	1·77
Fibrine per cent.,	0·047	0·0086

[1] *Beitr. Phys. Chem.*, v. 117.
[2] *Arch. gen.*, 1872.
[3] Halliburton, *Lancet*, July 28, 1890.

When withdrawn, the fluid may coagulate spontaneously.

This is not the rule, but rather the exception; and I believe it is an indication of unusually acute inflammatory mischief, and of a probably tubercular origin; so that, although the fluid may rapidly disappear and the pleurisy recover, the patient may soon prove to be the victim of tubercle, which may develop and progress very actively.

Among the organic substances sometimes found are urea, uric acid, occasionally sugar in diabetic cases, and biliverdin in jaundice. Glycogen also and cholesterin, and other fatty substances, occasionally occur, but these, I believe, only in long-standing cases. The presence of these substances is interesting, but of no practical importance.

Effusions may form with very great rapidity; thus they may become considerable in five to thirty hours after experimental irritation of the pleura in animals, and similar cases of rapid effusion are not at all uncommon in man.

THE EFFECT OF PLEURISY UPON THE ADJACENT ORGANS.—Pleurisy, whatever its origin may be, is likely to lead to changes in the organs adjacent, (1) as the result of the direct extension of the inflammation from the pleura to those organs, or (2) as the result of compression if a large effusion forms.

Of these organs, of course, the most important are the lungs; but the effect upon the diaphragm, intercostal muscles, pericardium, and mediastinum has also to be considered.

1. The spreading of Inflammation from the Pleura to the Lung Tissue.

The close connection of the connective tissue and lymphatic system of the pleura and subpleural tissue with that of the lungs, and the course of the lymphatic circulation from the pleura to the root of the lung, make it probable that the effect of inflammation would extend to the subjacent parts of the lung. Accordingly, in acute pleurisy we find the lymphatics of the lung infiltrated with cells, and containing an excess of lymphatic fluid. In some cases even the vesicles themselves may become involved, and superficial pneumonia be produced. If this does not resolve completely when the pleurisy subsides, development of connective tissue may occur, and so chronic superficial pneumonia be produced, which may extend some distance into the lung. This is the *chronic pleurogenic pneumonia of Charcot*.

This change is more likely to be met with when the lung has been long collapsed by effusion, and the pulmonary pleura has become thickened. In these cases the pleural thickening follows especially the interlobar septa and the coarse trabeculæ of the lung, although the finer trabeculæ may also be involved.

2. The consequences of Effusion—

1. *Upon the lung of the affected side.*—As effusion forms, the lung contracts by virtue of its own elasticity, thus driving the air out of itself; and when a large effusion has formed, the whole lung may pass into a condition of collapse (atelectasis), which may be so complete that the lung may look like fœtal lung and sink in water. Even when so completely collapsed as this, the lung may still be capable of re-expansion as soon as the fluid is removed, and it may not even have lost its elasticity, or at any rate not entirely; sometimes this is the case even after the lung has been collapsed for a long period, even for many months. In many cases, however, where the lung has been long collapsed, it becomes incapable of re-expansion, owing to the connective tissue changes which have taken in the pleura covering it, and in the trabeculæ of the lung itself.

Carnified Lung.—When the effusion is very large and the pressure considerable, the blood may be driven out of the lung as well as the air. To this condition the name *carnified lung* has been given to distinguish it from a collapsed lung, in which the air only has been driven out.

A carnified lung is of a slaty-gray colour, and not pink or red as a collapsed lung is; but no doubt many of the carnified lungs have also a good deal of connective tissue change in them as well.

When the effusion is not great, the resulting changes in the lung are local, so that only the parts immediately adjacent to the effusion are collapsed, while the rest is not only not collapsed at all, but may be in a condition of hyperdistension, the so-called *complementary emphysema.*

This local atelectasis is met with, not with effusion only, but wherever the cavity of the thorax is encroached upon. Thus it is seen in the neighbourhood of an intrathoracic tumour, an aneurysm, or an enlarged heart, or when the diaphragm is thrust up into the chest by ascites or abdominal tumour. Fagge states these facts in the form of a general law, viz., that whenever any part of the lung ceases to be acted on by the respiratory forces, it becomes collapsed, and that too even when the tubes are patent.

2. *Upon the diaphragm.*—When there is much effusion in the pleural cavity, the mere weight of the fluid flattens the diaphragm, and, if the quantity be large and the intra-thoracic pressure much increased, the diaphragm may be thrust into the abdomen, and its upper surface become concave instead of convex.

The effect of such distension as this is, first, to paralyse the muscle by stretching; and secondly, if the distension be long continued, to produce an actual degeneration and atrophy of the muscular fibres, so that the diaphragm may become thin and yellowish in colour, just as the abdominal muscles do in a case of chronic ascites. These changes in the diaphragm take their share in producing the dyspnœa present in such cases. However, even in such extreme cases, relief of the distension may in time be followed by recovery of the muscle.

When the pleurisy is inflammatory, still more important changes in the diaphragm are produced, viz., those which are the consequence of the extension of inflammation from the pleura to the tissue of the diaphragm itself.

The diaphragm is but a thin sheet of muscle intersected by connective tissue, strands, and lymphatics, and covered on both sides with serous membrane, the pleura and peritoneum respectively.

Inflammation, therefore, will easily spread not only into the diaphragm, but through it from one surface to another; and so we often find in association with diaphragmatic pleurisy a corresponding local peritonitis and *vice versâ.* In acute pleurisy the lymphatics are infiltrated with cells, and the muscle tissue either in a condition of inflammation or degeneration. If the pleurisy become chronic, the changes consequent on it in the diaphragm are likely to become chronic also, and an interstitial development of connective tissue take place, which subsequently shrinks, and in contracting compresses the soft muscle fibres and leads to their degeneration and atrophy. In extreme cases almost every trace of muscle tissue may have disappeared and microscopic examination show nothing but connective tissue.

Such extreme changes in the diaphragm are most frequently met with when the diaphragm has become adherent to the lung, and then the connective tissue may be half an inch or more in thickness.

These changes are often described as *acute* and *chronic Phrenitis.* The effect which such changes must necessarily have upon the function of the diaphragm is obvious, and in acute diaphragmatic pleurisy the consequent phrenitis is one of the chief dangers.

3. *Upon the intercostal muscles.*—The effects upon the intercostal muscles are the same in kind and produced in the same way. Thus it is the impaired nutrition of the muscles which accounts in great part for the bulging which takes

place with effusion. This bulging is most frequent and most marked with purulent effusion, but it is occasionally seen in serous effusions, and may even be local, *i.e.*. limited to that part of the side where the inflammation is most intense.

In chronic pleurisy the connective tissue change involves also the intercostal muscles, and assists in producing the immobility and contraction of the side which occurs.

4. *Upon the Mediastinum.*—A large effusion causes displacement of the mediastinum and the organs in it, but these more or less mechanical results of effusion will be dealt with in another place; here it is necessary to consider only the results which follow the spreading of inflammation. Thus the pericardium may become involved, and it is remarkable, considering the close connection that exists between the two structures, that pericarditis is not a more common sequel of pleuritis than it really is. Pleurisy seems more prone to spread to the mediastinal tissues outside the pericardium, and to the corresponding part above the pericardium, beneath the manubrium sternum. In the former case it may cause mediastino-pericarditis, and generally ends in undue fixation of the pericardium to the sternum, though if no pericarditis ensue, the consequence of this may not be evident or important. If in empyema suppuration spread to this tissue, it may lead to a mediastinal abscess, but this is fortunately a very rare complication. In the upper part of the mediastinum what commonly happens is a fixation of the pleura in the position in which it happens to be at the time, with some fibroid thickening of the tissues there, but this again commonly leads to no physical signs. If connective tissue developed to any great extent in this region, it might surround some of the large vessels there and cause obstruction to them. Such a result is a very rare sequel of pleurisy.

I have only seen one case, and that a very remarkable one, under the care of my colleague, Dr. Sainsbury,[1] in which the vena cava superior was completely obliterated by fibrous tissue which surrounded it, for which no other explanation could be found than an antecedent pleurisy.

5. *Upon the opposite lung.*—Except where there is a large effusion, no pathological changes consequent on pleurisy occur in the opposite lung.

When, however, the effusion is large, and one lung completely collapsed, the opposite lung is also partially collapsed as the result of the displacement of the mediastinum. It is also congested, because all the blood of the body has to pass through this one lung instead of two. The congestion shows itself during life by the signs of bronchitis, and after death by general vascular engorgement of the lung, most evident in the bronchi and in the lower lobes; the bronchi presenting the lesions of bronchitis and the lower lobes those of hypostatic congestion.

PATHOLOGICAL RESULTS.—When the inflammation subsides, the fluid may be absorbed; the false membrane disintegrate, undergo fatty degeneration, and be also absorbed; the pleuritic thickening and vegetations subside, and ultimately completely disappear.

Usually, however, the changes in the pleura persist to some extent, so that recovery is not complete; some thickening remains, and the inflammatory tissue becomes organised into connective tissue, which undergoes contraction.

If the pleurisy be limited to the visceral layer, the surface of the lung then shows a puckered cicatrix or a thin plate of cartilage-like texture, in which, after a time, lime salts may be deposited, and thus a thin calcareous plate be formed.

[1] *Lancet*, Nov. 30, 1895.

Usually the opposed surfaces of the pleura adhere, and the two surfaces thus become firmly united by connective tissue.

When these adhesions are extensive, and involve a considerable portion of the side, they draw the ribs close together, thus diminishing the intercostal spaces and depressing the ribs, so that the side is flattened and the intercostal spaces retracted, and in consequence the movements greatly diminished. In these conditions the intercostal muscles are found wasted, partly as a consequence of disuse, and partly because of an atrophy produced by the interstitial inflammation.

The **mechanism of the removal of fluid** is interesting.

The fluid may be removed in part, of course, by absorption through the blood vessels, but the chief factor in its removal is the pump-like action of the respiratory movements and lymphatic valves through the stomata on the surface of the lung and diaphragm. Hence it follows, that when the pulmonary pleura is covered with a thick membrane, so that the stomata are plugged; or when the lungs are partly collapsed, so that the stomata cannot open; or where, owing to extreme distension of the side, the respiratory movements are absent, the pump ceases to work, and the fluid can no longer be removed in this way.

The effects of the respiratory movements are shown by the results of paracentesis, for it often happens that the removal of but a small amount of fluid tends to the rapid disappearance of the rest; the explanation being, that the removal even of this small quantity of fluid has enabled the lung to expand somewhat, and permitted the respiratory movements of the side to commence again. In this way the pump is started to work once more, and slight though the effect may be at first, it is a constantly-increasing one, for every small amount of fluid removed renders the pump more effective, and so the disappearance of the fluid goes on in an ever-increasing ratio. When the stomata are permanently plugged, or the lymphatics obstructed by thickening in the pleura itself, the action of the pump can never be restored, and the effusion becomes permanent, unless after removal by paracentesis it should not again be exuded.

THE ETIOLOGY OF DRY PLEURISY AND PLEURISY WITH SEROUS EFFUSION.—In discussing the etiology of inflammatory affections of the pleura, it is necessary to keep the different forms of it distinct from one another. Many of the old statistics are inaccurate, because they are based on the assumption that dry pleurisy, pleurisy with serous effusion, and empyema are merely different stages of the same affection, and pass by easy gradations one into the other. We now know that empyema at any rate differs from the other forms, both in cause and course, and should therefore be kept clearly distinct. It is of course true that the same disease, for instance tubercle or pneumonia, may produce a serous and sometimes a purulent effusion; but this fact does not prove the identity of the two conditions, and must be set against those other facts which prove the difference between them.

Frequency.—In the Registrar-General's Reports pleurisy appears as the fifth in order of the causes of death among the whole population; but this statement is not very informing, because the different forms of pleurisy are not kept sufficiently distinct from one another, and the returns are necessarily not always explicit, for the death might be returned as one of pleurisy, and the real disease have been tubercle or cancer, with pleurisy as a complication, and *vice versâ*.

Hospital statistics, again, though more satisfactory, are still incomplete, for they are based to a great extent upon the in-patients, *i.e.*, upon the more serious cases, and though these statistics would give some indication of the frequency of the severe cases, they would give no true measure of the real frequency of the diseases in question, or of the relative importance and frequency of severe cases among them; for example, probably every case of empyema which applies to the hospital and is recognised is admitted, as well as all cases of dry pleurisy, in which the symptoms are severe, and all cases of pleuritic effusion in which the quantity of fluid is large; while a large number of cases of small effusion or of dry pleurisy, in which the symptoms are not severe, would be treated as out-patients, and would not come into the statistics at all.

Bearing in mind these necessary qualifications, the following statistics, taken from the records of St. Bartholomew's Hospital for ten consecutive years, are of interest; but here, again, it must be remembered, that only those cases are recorded as pleurisy in which the pleurisy is the primary or most important affection, and but few, if any, of the cases in which pleurisy occurs as a complication in the course of other diseases would enter into these figures.

In the ten years, 1883 to 1892 inclusive, there were admitted 638 cases of pleurisy, of which 238 were dry pleurisy and the remainder, 400, were pleurisy with serous effusion.

This was out of a total number of medical cases of 23,898, yielding a percentage of 2·7 per cent. as representing the frequency of the disease among in-patients.

Of these, 7 per cent. died, yielding a percentage of 1 out of the total medical deaths in the hospital.

During the same period there were admitted 227 cases of empyema, with 50 deaths, yielding a percentage of 0·9 of cases admitted, and 1·15 per cent. of the total deaths.

The actual frequency of pleurisy among sick people attending at the hospital would be more nearly arrived at if to these figures were added the number of cases which presented themselves in the out-patient room, and this appears to be about half as many again, *i.e.*, for every three which were treated as an out-patient two were admitted into the hospital.

I do not know that any conclusion of practical importance can be drawn from these or similar figures.

Mortality.—The general mortality of the two forms of pleurisy together among the cases admitted into the hospital is 7 per cent. Nor is there any great difference between the mortality of the two forms. If any, it is higher somewhat in dry pleurisy, viz., 7·6 per cent. as against 6·3.

The side affected.—It has been stated that pleurisy is more common on the right side than on the left, even in the proportion of 2 or 3 to 1; but the large statistics collated by Wilson Fox show no material difference, for right-sided pleurisy was found in 45·7 per cent., left in 49·9 per cent., and double pleurisy in 4·4 per cent.

My own figures, for a small number of cases, yielded the following result:—

Of 200 cases, 96 were on the right side and 104 on the left.

Age.—Fifty per cent. of all the cases of pleurisy, as the tables show, occur between the ages of 20 and 40, at the period, that is to say, at which phthisis is most prevalent. This is a fact of some interest in respect of the relation in which pleurisy stands to tubercle.

No age, however, is exempt, and though among the old and the young the frequency is smallest, the mortality, as might be expected, is highest. Even in the infant pleurisy is known to occur, and when met with immediately after birth, it is probably of septic origin. It has also been recorded in a fœtus whose mother had, shortly before parturition, suffered from an attack of fever.

Sex.—Sex is a strongly predisposing factor; thus pleurisy is far more common in males than in females, even in the proportion of 2 or 3 to 1.

My own statistics yield about 2½ to 1—456 males to 182 females.

This relation holds throughout life for all age periods, and cannot, therefore, be simply referred to the greater exposure of the male sex. It holds even in young children, but in infants it is said that the difference does not exist, and that the sexes are equally liable: but in the statistics for infants and children the numbers are small and the conclusions vary.

Season and chill.—The general seasonal curve of pleurisy agrees closely with that of pneumonia, for both affections are most prevalent at the breaks of the year, that is, in the spring and late autumn.

The seasonal distribution is given by Ziemssen[1] as follows:—

Dec.,	222	28·18 per cent.	June,	277	25·74 per cent.
Jan.,	387		July,	254	
Feb.,	247		Aug.,	251	
Mar.,	297	28·37 per cent.	Sept.,	173	17·71 per cent.
Apr.,	293		Oct.,	162	
May,	272		Nov.,	203	

A history of exposure to cold and wet, with consequent chill, is so frequently given by patients as the cause of their illness, that it cannot be disregarded as an etiological factor, although we may have no clear conception how it acts. Even in diseases of bacterial origin, exposure and chill play an important part in determining the attack. And again, after convalescence from acute diseases, such as typhoid, scarlet fever, diphtheria or influenza, when pleurisy occurs, it is often referred, and apparently correctly, to a chill; yet in some of these cases the specific organisms have actually been discovered in the pleura. I suppose we must conclude that by the chill the vitality of the tissues has been lowered, so that they cease to offer their normal resistance, and become an easy prey to the specific organisms.

Exposure, no doubt, explains in part the prevalence of pleurisy among males; but, as already stated, the sexual difference holds throughout the whole of life, and therefore the greater exposure to which the male is subject cannot be the complete explanation of the sexual difference.

Extension of inflammation from the parts adjacent.—If the adjacent inflammation be suppurative, the secondary inflammation in the pleura will probably be suppurative too; but not necessarily, for the pleurisy may be dry and adhesive, or if there be effusion, it may be of serous character.

1. From the lung itself.—As the result of any inflammation in it, for instance, pneumonia, broncho-pneumonia, infarct, abscess, and gangrene.

(*a*) *Pneumonia.*—Some degree of pleurisy never fails when pneumonia reaches the surface of the lung. Usually this is a dry pleurisy, and rapidly disappears when the pneumonia subsides. Occasionally fluid forms, and as the pneumonia resolves, becomes the prominent affection. The fluid may be either serous or purulent, but the latter much more frequently than the former, in the proportion of 3 or 4 to 1. When it is purulent it is often described by the term "metapneumonic empyema," and it will be discussed under this heading later.

In either case the pleurisy is probably bacterial in origin.

During the acute stage of the pneumonia the pneumococcus is commonly found in the effusion, but after the crisis the fluid is usually sterile, or at any rate the pneumococcus is very difficult to discover.

[1] *Ætiolog. der Pleur.*, 1889.

The pneumococcus may produce pleurisy without any obvious pneumonia.

(*b*) *Broncho-pneumonia.*—Broncho-pneumonia is much less frequently a cause of pleurisy, unless the broncho-pneumonia be the result of some specific disease, like scarlet fever, whooping-cough or diphtheria; but if it be of tubercular origin, pleurisy, in one form or the other, is hardly ever absent.

(*c*) *Infarcts.*—Extensive infarcts may exist in the lung without any pleurisy at all. If they are septic in nature, pleurisy is the rule, and it is usually of the suppurative kind.

The same holds of abscess of the lung or gangrene, or any other septic inflammation.

2. From the chest walls.—Any inflammatory affection of the chest walls is liable to spread through them and involve the pleura, although this does not happen as commonly as might be anticipated. Thus, pleurisy may follow an abscess of the chest walls, caries of the ribs, gumma, as well as new-growths.

Affections of the breast, again, are not nearly so frequently the cause of pleurisy as might be expected, for even very extensive inflammation or abscess may exist without the pleura becoming involved at all. So also with new-growths, and it is in either case only when the tissues beneath the breast are involved that the inflammation is at all likely to pass through to the pleura.

In women who are suckling, though in otherwise good health, I have occasionally observed pleurisy develop in the region of the breast, and where the patients suckle with one breast only, on that side. This may be referred to a chill consequent on the frequent exposure of the breast during suckling, but I do not think this a likely explanation, considering the thickness of the covering which the suckling breast provides. It is, I think, rather to be connected with the general activity of the circulation through the breast and through the vessels of the chest-walls connected with it.

3. From the parts within the thorax.

(*a*) *From the pericardium.*—Close as the connection between the pleura and pericardium is, it is comparatively rare for inflammation of the one to lead to inflammation of the other.

When this occurs, it is much more common for the pleura to become affected in consequence of pericarditis than *vice versâ*.

Pleuro-pericardial friction is always of interest on account of the difficulties of diagnosis, but the inflammation is frequently found to be limited to the pleura, even when that peculiar form of friction has been well marked.

(*b*) *From the mediastinum.*—Inflammation of the mediastinum is comparatively rare, and when it is met with, more frequently follows pleurisy than precedes it.

With new-growth and sometimes with aneurysm in the mediastinum, pleurisy may be associated. It is often of the dry adhesive kind, with much infiltration and thickening.

When effusion occurs, it is more often of the nature of dropsy rather than of inflammation, and is due to mechanical obstruction to the circulation through the veins.

(*c*) *From the diaphragm.*—Inflammation of the pleura and of the peritoneum are often consequent, the one on the other, owing, in the first place, to the thinness of the diaphragm, and secondly, to the close communication through the diaphragm between the lymphatics of each cavity. Thus chronic peritonitis is often found associated with chronic pleurisy, spreading from the diaphragm upwards. With acute peritonitis near the diaphragm, especially if it be of a suppurative or septic character, pleurisy is very common. Thus it is likely to be associated with subphrenic abscess, with hepatic abscess, with abscess the result of gastric

ulcer, or with abscesses which have developed in lower parts of the abdomen and tracked upwards, as for instance abscesses in connection with the cæcum or vermiform appendix, or even in some cases of psoas abscess. In most cases the secondary inflammation of the pleura is, like that of the peritoneum, suppurative, but not necessarily so, for it may be adhesive, or lead to serous effusion.

(*d*) *From the spine.*—Here the two common diseases are caries of the bones and new-growth, the former leading as a rule to empyema, the latter frequently to serous effusion.

Traumatic causes.—Pleurisy may follow a blow on the chest, but usually it is secondary to some injury done to the chest walls or lung ; yet the ribs may be extensively fractured, and the lung extensively contused, without any pleurisy following at all.

Where the chest walls have been punctured, as by a stab, other causes probably come into play, and the pleura will, in all probability, have been infected at the time. If the instrument be clean and no infection occur, the lung may be most extensively wounded and even considerable hæmorrhage occur, either from the lung or into the pleura, without any inflammation.

Bright's disease.—With *acute Bright's disease* serous effusions are more common than purulent, and they are, for the most part, dropsical rather than inflammatory, and are generally associated with other signs of general dropsy. The same is true also of *amyloid disease.* With *granular kidney* inflammation of the pleura is, I think, more common ; it is usually of the dry variety, but is sometimes attended with serous effusion. It does not, I think, occur often, except in the later stages of the disease, and is a somewhat grave complication, though it does not possess the gravity which acute pericarditis, under these circumstances, does.

Dickinson[1] gives the following statistics of the frequency of pleurisy and hydrothorax in tubular nephritis, granular kidney, and amyloid disease :—

	Number of cases.	Hydrothorax.	Pleurisy.
Tubular nephritis, ...	39	27 per cent.	20 per cent.
Granular kidney, ...	68	34 ,,	10 ,,
Amyloid disease, ...	48	2 ,,	10 ,,

With **gout,** pleurisy is not at all uncommon. With chronic gout the pleurisy is generally dry, and may stand in close relation to the chronic interstitial change in the kidney, with which gout is so often associated. In acute gout, dry pleurisy sometimes develops very suddenly and may almost as rapidly disappear, so that one is tempted to place the pleurisy in the same category as the metastases which occur from one joint to another. There are a few cases recorded, in which gouty deposits of urate of soda have been found on the pleura. I have, myself, seen several instances of acute pleurisy in the course of gout, and, so far, I should be inclined to agree with Garrod's statement that pleurisy is by no means uncommon in gout, although Duckworth's experience does not lead him to the same conclusion. Uric acid has been found in pleural effusions, but so it has in all other effusions, and I do not know that this is any proof of its gouty origin. In some cases of gout, dry pleurisy may attack the diaphragm, and cause a violent hiccough or spasmodic cough ; but this is fortunately a rare complication.

[1] *Pathol. and Treatment of Albuminuria.*

Specific diseases.—Pleurisy is by no means an uncommon complication in any of the acute specific fevers, excited, I think we may fairly presume, by the specific organism of the disease. Indeed, in some cases these organisms have been actually demonstrated, as, *e.g.*, in the case of pneumonia and typhoid.

In all diseases which are of a septic character, the inflammation of the pleura will in all probability be suppurative, though this does not necessarily follow.

Rheumatic Fever.—A good deal has been written recently about what is called "rheumatic pleurisy." This is an unfortunate term, for it lacks precision. It is often used by writers for cases in which pleurisy is associated with pains in the joints, or even in the limbs, without any other evidence that these are due to rheumatic fever. The term is also used sometimes to describe cases of pleurisy, in which no other cause can be found for the disease, except a chill or exposure. Such uses of the term are misleading, and "rheumatic pleurisy," if the term be used at all, should be restricted to those cases in which there is evidence that it is connected with rheumatic fever. As a complication of rheumatic fever, pleurisy is by no means common. The collective statistics given by Garrod yield a percentage only of 4·3. It is, for the most part, dry and of short duration. Occasionally effusion forms, but it is, without exception, serous in character, and runs a simple course.

A few instances are recorded in which serous effusion in the pleura has been the first manifestation of an attack of rheumatic fever, just as is sometimes seen in the case of an effusion into the pericardium.

Of the latter I have seen several instances, but of pleuritic effusion, as the initial symptom of rheumatic fever, I do not remember to have seen a single case, unless the following was an instance of the kind.

A lad of 14 years of age came under observation with an effusion on both sides. He had had what was apparently an attack of rheumatic fever, with joint pains as usual, one month previously. His present attack was recent, and might possibly have been a relapse. At any rate his temperature, which was 103 on admission, fell in three days to normal, just as it might in rheumatic fever. Paracentesis was performed on both sides, with the removal of 6 ounces of serum fluid from the right, and 36 ounces from the left pleura, and in the course of convalescence he was attacked with pericarditis.

A few instances are also recorded, in which a metastasis appears to have taken place from the joints to the pleura, and from the pleura elsewhere, just as we see occur from joint to joint, but these cases are extremely rare. In most cases, where pleurisy occurs in the course of rheumatic fever, it is found on the left side, and consecutive, in the great majority of cases, to pericarditis. Still there are cases of pleurisy in the course of rheumatic fever which cannot be referred to direct extension of inflammation from the pericardium.

In *gonorrhœal rheumatism* pleurisy is very rare, and is met with generally in those cases which, from their obstinacy and other symptoms, ought rather to be called chronic pyæmia.

Faitout gives a *resumé* of the subject, but although several cases are recorded of the association of pleurisy with gonorrhœa, the connection between them is a matter of opinion only.[1] In but few cases has bacteriological observation[2] supported that view. In one case Mazza found the gonococcus both by the microscope and by culture in the pleuritic fluid.

Syphilis.—Cases have been described under the term "syphilitic pleurisy." By this is meant pleurisy due to the organisms of syphilis, not a pleurisy secondary to gumma or syphilitic disease of the lungs, or of the walls of the chest. The existence of such a form of pleurisy is practically incapable of proof at present, and the mere recovery after treatment with anti-syphilitic remedies is inconclusive.

[1] *Arch. gén. de Méd.*, Oct. 1895.

[2] Mazza, *D. med. Woch.*, 1894, No. 22.

The Connection of Pleurisy and Tubercle.

I have reserved to the last in the discussion of the etiology of dry and serous pleurisy that part which is of the most importance and interest, viz., their relation to tubercle.

This question may be attacked from various sides, viz.,

1. *The history of the patient.*
   - (*a*) The hereditary antecedents or family history.
   - (*b*) The previous history.
   - (*c*) The subsequent history.
2. *The morbid anatomy.*
3. *The Examination of the fluid.*
   - (*a*) By the microscope.
   - (*b*) By cultivation experiments.
   - (*c*) By inoculation experiments.
4. The results of tuberculin injections.

1. The history of the patient.—In tuberculosis of the serous membranes hereditary taint plays a less important part than in other forms of tuberculosis;

Still Sittmann[1] found a history of inheritance in nearly 25 per cent. of his cases of pleural effusion.

Again, in a series of cases examined with reference to this point, 20 per cent. were found to have some obvious antecedent tubercular lesion, in the glands, or elsewhere, while a still larger number become tubercular subsequently.

Thus, of 92 cases which required paracentesis in the hospital at Dresden, Fiedler[2] states that 17 died of tubercle in the hospital, 8 more subsequently, and 66 were highly suspected to be tubercular: 21 only remained well at the end of two years. This gives a percentage of tuberculosis of 82. Barr's[3] experience at Leeds is much the same. Of his 62 cases, 3 died of tubercle in the hospital, and 18 within five years subsequently, yielding a percentage of 35·5. Bowditch[4] has published three sets of statistics: the first for ten years, yielding a percentage of 43·3; the second, also for ten years, yielding a percentage of 47; and a third series yields a percentage of 24; *in toto* giving an average percentage of 38.

Lemoine[5] found that of 28 cases, 5 only were apparently cured, 7 had doubtful signs of phthisis, 1 was tubercular at the time, and 15 became tubercular later.[6]

2. Morbid anatomy.—*Post-mortem* examination frequently reveals the existence of tubercle that was not suspected, both in the lungs and in the pleura, and so far confirms the conclusions to which the statistics just quoted point. Many statistics bearing on this point have been given in discussing phthisis. We may conclude that of all cases of pleurisy, at least 50 per cent., and possibly 75 per cent., are of tubercular nature.

3. The examination of the fluid.—The examination of the fluid for the bacillus by the microscope, as well as by cultivation, is in most cases negative, although recently Gravitz, after concentrating the effusion by centrifugal action, has been successful in both methods.

Inoculation, however, has been often successful when other methods have failed.

Thus, in 20 cases of apparently primary sero-fibrinous pleurisy, 8 proved by inoculation to be undoubtedly tubercular.[7]

[1] Charcot, *Traité de Méd.*, 978.
[2] *Volkmann's Samml.*, 1882.
[3] *Brit. Med. Jour.*, 1890, May 10.
[4] *Med. News*, 1889.
[5] *Bull. d. Hop.*, 1895, p. 256.
[6] Kelsch and Vaillard, *Arch. de Physiol.*, 1886, ii. 162, examined 16 fatal cases of acute pleurisy, and found tubercle in the pleura in all, and out of 113 clinical cases, 82 per cent. proved to be tubercular.
[7] Netter, *Soc. méd. des hôpitaux*, 1891, April 17.

At the same time, a negative result does not prove an effusion to be non-tubercular.

For instance, out of 7 cases of undoubtedly tubercular pleurisy, inoculation succeeded only in 2. In another series of experiments, of 5 apparently primary cases of pleuritic effusion, inoculation proved it to be tubercular in 2 ; while in 4 cases, which were certainly tubercular, inoculation was successful only in 1.

Chauffard and Gombault[1] examined 20 cases, 10 of which yielded evidence of tuberculosis on inoculation, though microscopically tubercle bacilli had not been found.

	Thue.[2]	*Fernie.*[3]
	10 cases out of 30, 20 being sterile.	14 out of 20, 6 being sterile.
Bacillus tuberculosis,	1	3
Streptococcus,	1	..
Staphylococcus albus,	3	6
Staphylococcus albus and aureus,	1	...
Other cocci,	4	4
Pneumococcus,	...	...
Typhoid bacillus,	...	1

4. The injection of tuberculin in pleuritic effusion.

Out of 15 cases, 13 gave a well-marked tubercular reaction, that is, 87 per cent., a percentage almost as high as that which is yielded as the result of tuberculin injection in patients who are notoriously tubercular.

Finally, Debove and Reynault[4] have shown the presence of tuberculin in serous effusion by its action upon lupus.

There is but one conclusion to be drawn from all these observations, viz., that tubercle is a much more common cause of dry and serous pleuritis than has been hitherto supposed.

With empyema the case is different, for the relation of empyema to tubercle is by no means so close.

60. DRY PLEURISY—PLEURITIS SICCA.

An acute pleurisy is called dry when the inflammation is unattended with the accumulation of fluid in the pleural cavity, and leads to no more than a certain amount of plastic exudation. This, in many cases, is not more than sufficient to produce some roughening of the surface, and therefore causes no alteration in the percussion-note.

In other cases, the exudation forms a jelly-like layer, which, as a rule, is not thick enough to cause more than slight impairment of the percussion-note, but it may occasionally be as much as half an inch in thickness, or more ; then the note will be dull, and the diagnosis from a local collection of fluid not altogether easy.

Dry pleurisy may be *acute* or *chronic*, *inflammatory* or *non-inflammatory*.

Acute dry pleurisy is usually inflammatory, and it is this form which is associated with the most acute symptoms. The non-inflammatory dry pleurisies are, for the most part, chronic, usually secondary to some chronic disease in the neighbourhood, and are often attended with few or very indefinite symptoms.

[1] *Bull. d. Soc. Méd.*, 1886, p. 441.
[2] *Bull. d. Hop.*, 1895, 439.
[3] *Ibid.*, 145. Kingston Fowler, 547.
[4] *Soc. méd. des hôpitaux*, 1891, July 24.

SYMPTOMS AND PHYSICAL SIGNS.—Acute inflammatory pleurisy generally sets in suddenly, with a severe stitch in the side, and some fever.

Pain in the side.—This is the most prominent symptom, and is rarely absent.

The pain is characteristic, being a sharp *stitch*, stabbing or cutting in character, causing an abrupt catch in the breath.

It is increased on breathing, speaking, or coughing, and sometimes by pressure. It is almost always relieved by restraining the movements of the side, as by strapping or bandaging. It is usually *localised*, *i.e.*, limited to the particular part where the lesion exists, and it may therefore be felt in any part of the chest.

The commonest *seat of pain* in simple dry pleurisy is the mid-axillary region in the fifth or sixth intercostal space; and this is so, even when the pleurisy is not limited to this region but is widely extended, involving, it may be, the whole base of the lung. No doubt this is in part to be explained by the fact that the movements of the side are freest here.

The seat of pain is often extremely tender to percussion, and there may be so much cutaneous hyperæsthesia that the slightest touch causes exquisite pain.

Sometimes the pain is *radiated* or *referred* to other places; thus in the upper part of the chest it may be referred to the shoulder, clavicle, or sternum; lower down to the groups of cutaneous branches of the intercostal nerves, in the mid-axilla, near the sternum in front of the spine behind; or sometimes it is referred to parts below the ribs altogether, *e.g.*, to the hypochondrium and epigastrium, or below these levels to the hepatic or umbilical region, to the lumbar region, or even, it is said, as far down as the crista ilei. This is especially the case in children, possibly because the abdomen is such a common seat of pain in them from other causes. In adults it is chiefly when the diaphragm is involved that the pain is referred to the abdomen, but I have met with this, even with pleurisy fairly high up in the thorax. With pleurisy on one side, the pain has been sometimes referred to the other, or felt in both.

Some dry pleurisies are not painful. In most of these cases the pleurisy is chronic; but pain is sometimes absent, even in acute pleurisy; especially in aged debilitated patients, and in lunatics, or when pleurisy occurs in the course of gout and granular kidney.

The pain is not necessarily worse in neurotic or nervous patients.

The pain is, no doubt, in most instances due to the rubbing of the pleural surfaces on each other during the movements of respiration, for it may be greatly relieved by controlling the movements of the side with a bandage or strapping.

It is stated to be often absent when the pulmonary pleura alone is affected, and it is certainly most severe when the parietal pleura is involved.

If the pain be localised, it is due either to the rubbing of the surfaces together, or to a local inflammation of the intercostal tissues. If radiated, it must be due to irritation of the intercostal or other nerves.

The pain may disappear if effusion takes place, and return when the effusion is removed. (*Redux pain.*)

In regard of the pain alone, dry pleurisy might be difficult to diagnose from rheumatism of the intercostal muscles and from intercostal neuralgia, *i.e.*, pleurodynia, but the occurrence of friction would be conclusive.

Friction.—As pain is the most striking symptom of dry pleurisy, so is friction the most characteristic physical sign. It is produced by the rubbing together of the two layers of the pleura, and is therefore heard only where the pleura is roughened and the movements fairly free, *i.e.*, over the seat of the lesion.

If the movements are greatly restricted, as they often are, the friction may not be audible at all until the patient coughs or takes a deep breath.

At first, and in slight cases, it may be sharply localised, but it may travel over a wide area as the pleurisy extends, and if fluid form it may disappear at the spot where it was first heard, but still be audible at the margins.

In acute cases it may be heard within an hour or two of the onset of the disease; it may persist for a long time, for days or even weeks. Sometimes it may be easily felt as well as heard, and the patients be even conscious of it themselves.

Attention is usually drawn to the seat of friction by the pain complained of, but it may be discovered on auscultation when there is nothing in the symptoms or the history to suggest its presence, *i.e.*, dry pleurisy may be latent, as other forms of pleurisy often are.

It may, of course, be heard in any part of the chest, but the commonest place for the friction as for the pain is the lower part of the side in the mid-axillary region.

Friction, in its most characteristic form, is a coarse rubbing to-and-fro, *i.e.*, double sound, synchronous with respiration, like that produced by rubbing two surfaces of dry leather together, and therefore described as the *dry leather creaking*. This, however, is not the only form of friction, nor even, perhaps, the commonest. In many cases the sound is more crepitant than creaking, so that it cannot be distinguished by the ear alone from superficial crepitation in the lung.

When the pleurisy is near to the heart, the friction sounds may be synchronous with the movements of the heart, and will be difficult to diagnose from pericardial friction. It is then called *pleuro-pericardial friction*. As a rule, pleuro-pericardial friction is distinctly modified by respiration. The best way to test this is to cause the patient to inspire as deeply as possible and hold the breath; then, after noting the characters of the friction, to cause the patient to expire as much as possible and again hold the breath, and compare the characters of the friction in the two phases.

Other Physical Signs.—*The movements* of the side are more or less restricted because of the pain. This is a reflex phenomenon, and one which cannot be intentionally imitated. I have even seen one-half of the diaphragm inhibited while the other still continued to contract.

The side is also often flattened as well as restricted in movement, an effect probably due to the spasmodic contraction of the muscles consequent on their irritation.

These changes in shape and movement are most easily detected by bimanual palpation, *i.e.*, by placing the two hands on the corresponding parts of the two sides; but they are often obvious enough to the eye.

As another result of the defective movement, less air will enter the corresponding portions of the lung, and *the breath-sounds* there be less distinctly audible. The respiratory murmur, though diminished, is not altered in character, *i.e.*, it still remains vesicular.

Vocal vibrations and *vocal resonance* remain unaffected unless the exudation be of some thickness, when they will both be somewhat diminished.

The *rate of breathing* is often greatly accelerated, for as the pain prevents the patient taking a deep breath, a number of shallow ones are taken instead. In most cases the pulse is not accelerated, or, at any rate, not much, so that an alteration is produced in the *pulse-respiration ratio*; and this may even be as marked as it is in pneumonia. This is often seen in children, but occurs, though not so frequently, also in adults. The same thing is seen in pericarditis, where there is much pain, and for the same reason, and if there has been no acceleration of respiration before, it is very likely to be produced if pericarditis develop as a complication of pleurisy.

Sometimes there is a short, dry, catchy *cough*, which may be frequent, even paroxysmal, and very distressing from the pain it causes. Fortunately, cough is generally absent, or at any rate not severe or frequent.

When pleurisy affects the diaphragm it may be attended with very troublesome *hiccough*. This I consider a grave sign, but fortunately it is also a rare one.

Decubitus.—The patient will lie, of course, in the position most comfortable to him, and what this will be it is impossible to foretell. As a rule, pressure on the affected side increases the pain, the patient does not lie upon it for that reason, but if pressure does not cause pain, the patient will prefer to lie upon the affected side, because the respiratory movements will be checked in it as they are when the side is strapped, and the other lung will be freer to move.

Fever.—The temperature is generally raised at first, but not very much. It rarely rises above 101 or 102, and does not remain at this height for more than a few days. It remits during the day, and there may be as much as a difference of 2 degrees between the morning and evening temperature, and it may not drop to the normal for a week or ten days. There is sometimes a difference in temperature upon the two sides, that of the affected side being $\frac{1}{2}$ to 2 degrees higher than the other. This is observed as often with pleuritic effusion as with dry pleurisy, but it is more often absent in either case than present, and is of no diagnostic or clinical importance.

During the first few days, while the temperature is at its highest and the symptoms most acute, there may be some general *constitutional disturbance*—the skin dry, the tongue coated, the appetite impaired; there may be headache, and constipation is common. In the course of a day or two, as the fever subsides, all these general symptoms pass off.

The condition of the *pulse* varies with the fever. It is rapid if the fever be high and the symptoms acute, and the tension is then usually increased. In the later stages it varies with the patient's general condition. When the fever is past the pulse becomes slow, and the tension falls to normal or, for a time, below normal.

ONSET.—The onset of acute dry pleurisy is generally sudden. The patient may feel chilly and shivery, but anything of the nature of a rigor is not common; if a rigor occur, the presumption would be rather in favour of pneumonia than of simple pleurisy.

The first symptom, as a rule, is the stitch in the side, associated with some fever and constitutional disturbance, but the general symptoms of pleurisy vary greatly, and in some cases there are no symptoms at all, so that the diagnosis has to be made by physical examination alone. This has been called *latent pleurisy*.

DURATION.—The duration of an attack of simple dry pleurisy is from ten days to a fortnight, at the end of which time the patient is practically well.

SEAT.—The common seat of idiopathic, *i.e.*, simple spontaneous dry pleurisy, is the lower part of the mid-axillary region, but if dry pleurisy be secondary to some other affection of the lungs or thoracic walls, *e.g.*, phthisis, pneumonia, a broken rib, etc., it might be found in any other part of the chest wherever the lesion happened to be.

DIAGNOSIS.—When the physical signs are well marked, the diagnosis is obvious, and the only question is that of the cause.

A. Of the Cause.—Dry pleurisy is so frequently an early symptom of tubercle, that the possibility of what appears to be a spontaneous pleurisy proving to be of tubercular origin has always to be borne in mind. Still there can be no doubt that dry pleurisy is frequently of a simple nature, and produced by simple causes, of which the chief is exposure or chill.

Of simple dry pleurisy the usual seat is the lower part of the mid-axillary regions.

When not in this position the probabilities of tubercular origin become much greater. Thus tubercle is probably the real cause of the affection—

1. If the pleurisy be at the apex.
2. If it occur in patches and be irregularly distributed over the side.
3. If it be general, over the whole lung.
4. And still more, if it be bilateral, for, in all these cases alike, its distribution suggests that it is not a primary pleurisy, but secondary to some lesion in the lung, and there is little else for this lesion to be except tubercle.

The frequency with which acute dry pleurisy is of tubercular origin is difficult to estimate, but the percentage of cases is probably very much greater than has been until recently supposed; while a tubercular origin is most probable where the dry pleurisy is sub-acute or chronic, and especially if it recur without any obvious cause.

B. From other Affections.—When the physical signs are indefinite, the diagnosis will have to be made from intercostal rheumatism, muscular strain, intercostal neuralgia, and local lesions in the thoracic walls.

In *rheumatism* of the intercostal muscles there will often be the presence of muscular rheumatism in other parts, or the history of it, to assist the diagnosis.

Muscular strain in most cases is the result of coughing. The pain is often in peculiar places, and usually only during the act of coughing. Its favourite seat is at or near to the costal arch, at the insertion of the abdominal muscles into the thorax, *i.e.*, lower down than the common seat of dry pleurisy in the mid-axilla.

Intercostal neuralgia.—In some cases the recurrence of the attacks of pain in paroxysms with intervals of freedom will give the diagnosis, but where the pain is constant the difficulties will be greater.

Intercostal neuralgia is pain in the area of the distribution of the intercostal nerves, and, of course, pleurisy is one of its causes, but where the signs of pleurisy are absent, the other causes must be sought for, *e.g.*, irritation of the nerve near the spine, by disease of the vertebræ or ribs, new-growth or aneurysm. In the absence of such lesions, intercostal neuralgia is rare, except as the result of a past or present Herpes Zoster.

Local lesions in the thoracic walls are usually obvious enough, and are not likely to cause difficulty if ordinary care be exercised.

TREATMENT OF ACUTE DRY PLEURISY.—During the first few days, while the temperature is high, the patient should be kept warm in bed. The diet should be light and easily digestible, especially where the tongue is coated and the appetite bad. It will be well in such a case to give a mild aperient and prescribe a diaphoretic mixture.

Counter-irritation over the seat of lesion will do good, *e.g.*, hot mustard poultices or lint freely sprinkled with spirits of camphor or turpentine; or the side may be rubbed night and morning with lin. terebinthinæ aceticum, or painted with iodine; and in some cases of sub-acute pleurisy a light fly-blister is found beneficial.

Where *pain* is severe, immediate relief will often be given by strapping the side firmly to prevent its movement on respiration, or by applying a few leeches over the seat of pain, and letting the bites bleed for a time under a warm poultice, upon which, if necessary, a little opium may be sprinkled.

If the *rest be broken* on account of pain, cough, or, still more, hiccough, a morphia injection should be given. Morphia can be used in these circumstances without any disadvantage, and with very great benefit; but for the stitch in the side it is not as useful as might be expected, nor does it give as immediate relief as either strapping or leeching.

If there be any *cutaneous hyper-æsthesia*, the tenderness may be relieved by brushing the part over with tincture of aconite, or applying a compress soaked in a 20 per cent. solution of cocaine.

During *convalescence* great care will be required lest the patient should get cold and thereby contract a relapse. After a time he should be sent away for change, should be well fed, and placed upon tonics.

Patients who have had acute pleurisy should be regarded as delicate and wanting careful treatment for some months to come; and, if possible, it might be wise to send them away into a warm climate for the following winter.

61. PLEURITIS EXUDATIVA.

(Pleurisy with Effusion.)

OF EFFUSION IN GENERAL.

If over the seat of pleurisy the percussion be found dull, the pathological condition will be that of either pleuritic thickening or pleuritic effusion.

Pleuritic thickening may occur in any part of the chest, but fluid, if free to move, *i.e.*, not confined by adhesion and therefore localised, will gravitate to the lowest parts; thus it is not uncommon to find an affection of the upper part of the lung, *e.g.*, pleuro-pneumonia of the upper lobe, followed by pleuritic effusion at the base.

The seat of dulness thus becomes of importance in diagnosis; dulness limited to the uppermost parts of the chest is hardly ever due to fluid, but almost always to pleuritic thickening; dulness limited to the middle region is most likely to be due to pleuritic thickening, but might be the result of a localised effusion; while the lower part of the chest, *i.e.*, the base behind, is the favourite seat of effusion, and that even if it be localised.

THE PHYSICAL SIGNS.—The signs of effusion fall into two groups —(1) those *in situ* which prove the presence of fluid; (2) those elsewhere which are the consequences of the effusion, viz., the displacement of organs.

Wherever fluid exists there will be *dulness on percussion.*

With a layer of fluid half an inch in thickness, the percussion will be greatly impaired, and with a layer an inch in thickness will be absolutely dull.

Over a large effusion the dulness has a peculiar stony character, with an entire absence of resonance, which is very suggestive, for it is hardly ever, if ever, met with under other conditions. *The sense of resistance* on percussion is also greatly increased, even more so than over solid lung.

The *vocal vibrations*, *vocal resonance*, and *breath sounds* are more or less diminished or completely absent, according to the amount of fluid. This is a general rule, to which, however, there are some exceptions; for in children commonly, and in adults sometimes, the voice- and breath-sounds may be increased even to the extent of bronchophony and bronchial breathing. This will be referred to more fully by-and-bye.

The amount of fluid, *i.e.*, the size of the effusion, varies, and with it also to some extent the physical signs; so that it will be convenient to consider, first, the signs of a small or moderate effusion, and then those of a large effusion.

A. THE PHYSICAL SIGNS OF A MODERATE EFFUSION.

The limits of the effusion are usually easily determined by **percussion,** and thus we are enabled to gauge its amount, *i.e.*, estimate its size.

When the fluid is encysted, the position and shape of the area of dulness will depend upon the adhesions round it. If there are no adhesions, so that the fluid is free to take its own course, the dulness is first met with at the base behind, *i.e.*, at the lowest part of the chest, whence it spreads gradually upward as the fluid increases, and extends farther towards the front.

The upper border, it is stated, forms a curved line, "*Damoiseau's curve*," highest at the angle of the scapula, and falling towards the front as well as towards the spine, thus forming an S-shaped figure. This curve is said to be only met with in moderate effusions, the line becoming straight and more or less horizontal as the effusion increases. Many authorities do not believe in this curve at all; and I must say I have failed to satisfy myself of its existence. It is at any rate of little practical importance. I agree with Fagge and Ferber in the opinion that the line of dulness in any given case depends largely upon the position which the patient has occupied; for instance, whether he has been in bed or not, and which side he lies upon most.

Alteration of percussion lines with position.—In cases of hydro-pneumothorax, *i.e.*, where air and fluid are present together in the pleura, it is very easy to make out the change of level, for this takes place quickly, as the patient changes his position from the recumbent to the sitting posture; but it is not by any means so easy to demonstrate this change when fluid alone exists in the pleura. A change, indeed, does take place, but in most cases only slowly. The explanation is to be found in the fact already mentioned, viz., that as fluid forms, although the lung no doubt to some extent undergoes general shrinkage from its own elasticity, still the collapse is most marked, and may be complete, in those portions of the lung which are immediately adjacent to the fluid, while the rest seems to be *pro tanto* dilated. In this way the fluid is to some extent held up, as it were, against the action of gravity, or at any rate the lung will not float as readily, or change its position as easily, as we might expect.

The shape of the Chest.—Over the seat of effusion, that is to say, in the lower part of the back and side as a rule, the chest is rounder and fuller, and the intercostal spaces wider and more resistant than on the opposite side. The distension or bulging, though usual, is not invariable, for the side may be flatter and actually smaller than the other, and the intercostal places closer together; and this, too, with a recent and even an increasing effusion of fairly large size.

The contraction of the side must then take place at the expense of the lung, which must be more collapsed than usual for the same amount of fluid.

The **movements** also are almost always much impaired, and may be completely absent.

Ægophony.—Ægophony has not the value once attached to it in the diagnosis of pleuritic effusion.

Ægophony is a peculiar kind of vocal resonance, in which the voice, transmitted to the ear through the chest walls, is of a high pitch, and has a peculiar, tremulous, quavering character like the bleating of a goat, from which the name was taken. It has also been compared to the voice of Punch, or to the voice of one speaking through the nose.

It used to be thought pathognomonic of fluid effusion in the pleura, and was said to be audible at the upper limit only of a localised effusion. Neither of these statements is correct, for it is far more often absent than present with a localised effusion. It occurs sometimes with other conditions, for example, thickening of the pleura without fluid, pneumonia, phthisical consolidation, and even in the inter-scapular spaces of perfectly healthy persons. Nor is it a fact that it marks the upper limit of the fluid; on the contrary, it is generally heard well within the area of dulness, and often some distance within it, and hardly ever anywhere except at the lower part of the chest behind, or in the axilla, its favourite seat being near the angle of the scapula. Usually the area over which it is audible is limited in extent and quite small, but with a large effusion it may be considerable, and measure many square inches, but even then it is heard over the lower half of the back only.

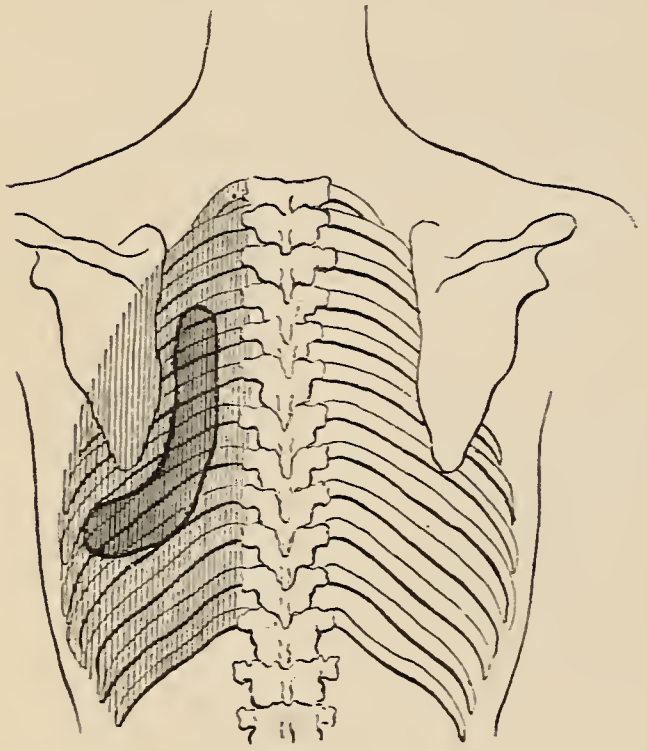


Fig. 145.

Diagram showing the peculiar distribution of ægophony over a large area in a case of very large serous effusion. The area measured 8 inches by 3 inches.

Ægophony thus ceases to be of any practical value in the diagnosis of fluid, but it is an interesting and striking phenomenon.

There is great difference of opinion as to the way in which it is produced. Laennec, to whom the original description is due, attributed it to the flattening of the air-tubes by compression, aided by a thin layer of fluid which could be set in movement by the vibrations of the voice.

The general view now held is that fluid in the pleura is mainly responsible for the production of the phenomenon, and that it does this either by intercepting the fundamental note or lower harmonics of the voice, or by supplying additional, discordant, secondary vibrations to it (Douglas Powell), or else by multiplying the media through which the vibrations of the voice have to pass (Fagge, Bristowe, etc.).

Dr. F. Taylor suggests that it is due to the reduction in length of the bronchial tubes by compression, so that they cease to vibrate to the lower fundamental tones or lower harmonics, and resonate only to the higher harmonics, thus producing a discordant sound.

B. THE PHYSICAL SIGNS OF A LARGE EFFUSION.

With large effusions the physical signs are the same in kind, though greater in degree.

Inspection.—The side is almost invariably distended, the angles rounded, the intercostal spaces widely separated, tense, and often prominent. The measuring tape may show a difference of an inch or so as compared with the opposite side, although that also undergoes some slight enlargement, perhaps to the extent of a quarter of an inch above its normal size. The difference in shape and size may be demonstrated by the cyrtometer tracing, but often not as clearly as inspection of the chest would lead us to expect.

The distension of the side is not invariably present. I have seen the side even become smaller and somewhat flattened, and that, too, while the physical signs showed that the effusion was still increasing. I was inclined to think, in this case, that the effusion could not be very large, yet I removed 150 ozs. of serous fluid without aspiration.

Signe de cordeau.—The distension, when extreme, causes slight displacement or twisting of the sternum. The xiphoid cartilage, under ordinary circumstances, stands in the middle line, that is to say, a thread stretched from the middle of the episternal notch to the middle of the pubes crosses the point of the xiphoid cartilage; but where effusion exists, and the side is greatly distended, the point of the xiphoid cartilage lies to the opposite side of this line. This is called by Pitres "*le signe de cordeau.*" It is most obvious in children, in whom the bones and cartilages are soft.

If the effusion be considerable, the *superficial veins* of the side may be considerably dilated, the pressure within the thorax causing the blood to return by means of the anastomatic channels with the subcutaneous veins. The dilatation may be sometimes observed to vary with respiration, the veins becoming more distended on expiration and less so on inspiration.

Dilatation of the superficial veins is rarely met with, except in effusions of long standing, and with high intra-thoracic pressure, and it is more common with purulent than with serous effusion, but in neither is it so marked or so frequent as in hydro- or pyo-pneumothorax.

There may be some *œdema* of the side; this, of course, is common enough when an empyema is pointing. Though it has been recorded as occurring also with serous effusion, I have never seen it myself, and I am sure it is very rare.

The movements of the side are greatly diminished, or even entirely absent, the chest remaining constantly in a condition of extreme distension; in fact, the extreme distension, associated with immobility of the side, makes the diagnosis very probable by inspection alone.

Palpation.—The vocal vibrations are usually entirely absent over the area of effusion, and that even when a certain amount of vocal resonance is retained.

In the intercostal space behind, *i.e.*, over the root of the lungs, round which, in a large effusion, the lung is collapsed, the vocal vibrations are generally distinctly felt, and are sometimes greatly exaggerated.

Percussion.—The dulness everywhere is absolute, except, it may be, at the clavicle or just above it. This region is at any rate the last part of the side to become dull; partly because, as the patient lies, it is the highest part of the pleural cavity and the lung naturally floats up there as long as it can; partly because, in so many cases, the lung is attached there by adhesions.

Displacement of Organs.—The pleura is distended to the utmost, and all the organs in relation with it are displaced.

The heart is carried far to the opposite side; the diaphragm is depressed, taking with it the organs in relation with it, viz., the liver on the right side and the stomach and spleen on the left. The position of these organs may be easily determined in the thorax by percussion, and in the abdomen by percussion, assisted by palpation.

With effusion on the left side the line of dulness starts from the left sterno-clavicular joint, and extends across the sternum to the right, reaching to an inch and a half or so from its right border, opposite the third rib, and thence passes in a slanting direction through or outside the nipple, to join the line of hepatic dulness, which remains still in its normal place. The heart is seen and felt beating in the right nipple line or outside it, and also probably in the epigastrium too; the apex-beat cannot be felt, as the apex of the heart lies beneath the sternum.

The area of stomach resonance is absent, so that the dulness reaches to the costal arch or very nearly so.

This is not quite constant. I have seen the stomach resonance present, even with a large left-sided effusion. This is generally owing, I believe, to distension of the stomach from flatus, probably dependent upon the taking of large quantities of milk.

The spleen is felt below the ribs, and it is said that sometimes the diaphragm itself can be felt protruding into the abdominal cavity below the ribs, but I have never been able to satisfy myself of this.

The left lobe of the liver is somewhat thrust down and compressed, but this does not make any difference in the normal lines of hepatic dulness, but we know that it is so displaced only by *post-mortem* observation.

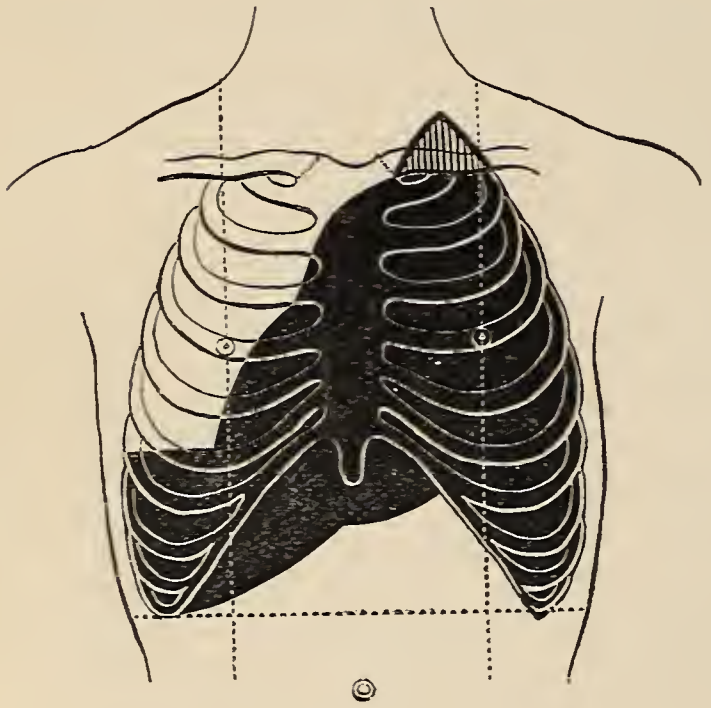


Fig. 146.

Diagram showing the displacements produced by a large effusion into the left pleural cavity.

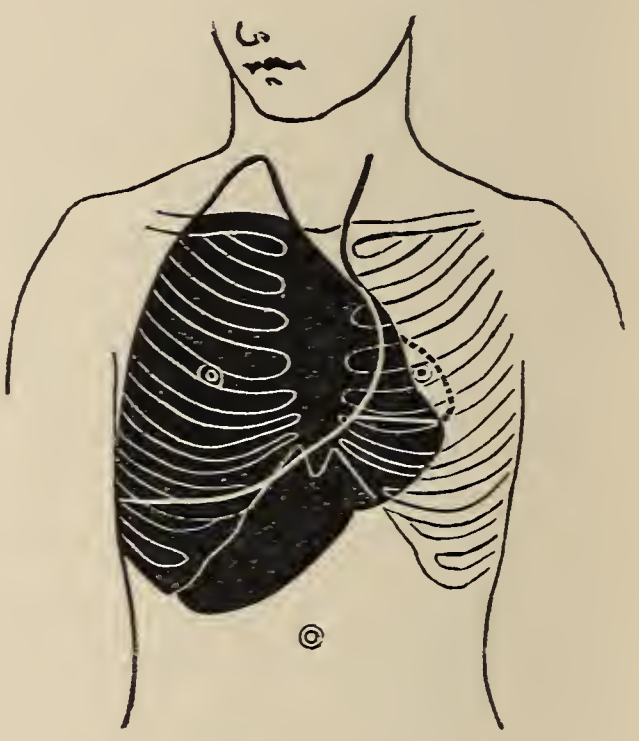


Fig. 147.

Diagram showing the displacements produced by a large effusion into the right pleural cavity. The white lines indicate the probable position of the upper border of the liver on the right side, and of the edge of the right pleura on the left side.

With effusion on the right side, the pleura extends across the sternum in the same way into the left side, the line of dulness being continued through the nipple, or above and outside it, to the spot where the apex of the heart is felt beating in the fifth or sixth space, an inch or more outside the left nipple line.

The stomach resonance is but little altered, and the spleen cannot usually be felt below the ribs.

The most marked change on this side is in the position of the liver, which is greatly displaced and curiously rotated.

The lower border reaches down as far as the level of the umbilicus, or even lower, 3 or 4 inches below the costal arch; the notch is then usually close under the left costal arch, and the gall-bladder in the middle line. The displacement and rotation of the liver is the same as that met with in pneumothorax of the right side.

Resonance beneath the upper part of the sternum, with a large effusion, is very rare, but I have seen one case in which the percussion was tympanitic here, yet 112 ounces were removed from the pleura. Possibly the lung was adherent, and thus prevented from collapsing.

Displacement of the Heart and its Consequences.—When the heart is displaced it is felt beating on the right or left side, as the case may be. On the left side this impulse corresponds with the apex of the heart; and on the right side, it was said to do so too, the heart being, it was supposed, fixed at its base by the large vessels, so that when it was displaced the apex swung over, and thus came to present on the right side, even beneath the nipple or beyond it.

How this theory became current it is hard to say, for the simplest pathological observation proves it to be untrue.

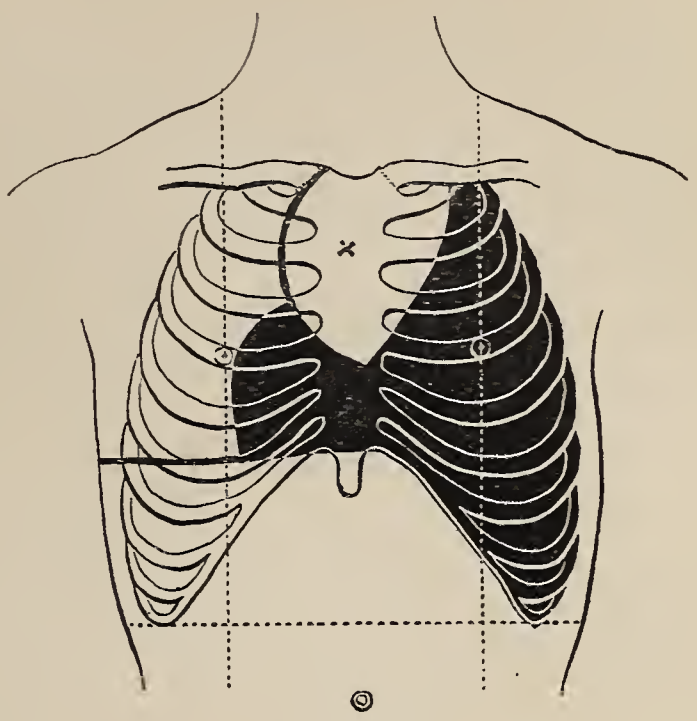


Fig. 148.

Large pleuritic effusion, with resonance beneath upper part of sternum (×).

Post-mortem observation shows that when the heart is displaced, it simply moves over from one side to the other, the apex being always towards the left, and that even with the maximum displacement to the right the apex does not reach beyond the sternum, lying as it does close to the xiphoid cartilage. *Cf.* fig. 122.

Extreme displacement of the heart, it might be thought, would be likely to have some effect either upon the action of the heart itself or upon the circulation through the vessels; yet it is remarkable how little effect it really seems to have. The right side of the heart is, it is true, sometimes found dilated; but for this there is a sufficient explanation in the difficulties of circulation through the lungs, without necessarily referring it to the displacement, and even with the maximum displacement it is often absent.

If, in extreme cases, the fluid does exercise pressure upon the heart, it does not appear to cause any embarrassment in its action.

Displacement, it might be thought, would be more likely to affect the vessels than the heart itself. Thus at the base the vessels might be stretched by displacement or compressed by the effusion, but there is no evidence that, if this occurs at all, it is sufficient to produce any obstruction to the circulation; or again, the vena cava inferior might be stretched or kinked as it passes through its orifice in the diaphragm, and thus the circulation through it rendered difficult.

Kinking of the vena cava has indeed been given as the explanation of the sudden death which sometimes occurs in pleurisy, and which was regarded by Moxon and others as a real and great risk in large effusions. In opposition to this theory, it must be stated in the first place, that sudden death is very rare in pleuritic effusion, and when it occurs, may be more correctly attributed to other causes; and in the next place, that though attention has been especially directed to this point on *post-mortem* examination, kinking of the vena cava has hardly ever been capable of demonstration—in other words, it does not exist.

Murmurs, it might be supposed, would very likely be produced by displacement. They do occur, but are by no means so common as might be anticipated. They are almost invariably systolic in time, and heard over the body of the heart as well as at the base.

At the base they are probably due to stretching and narrowing of the vessels by the displacement, or to their compression by the fluid, but of this they are the only evidence. Over the heart they are more likely to be connected with dilatation of the auricles or ventricles than with valvular incompetence. To justify the referring of a murmur to mere displacement of the heart, the murmur must not only be present when the heart is displaced, and disappear when the displacement is removed, but there must also be no more simple or ordinary explanation to account for it. Murmurs which answer to these requirements are very rare with effusion. I have only seen one or two instances myself.

Displacement of the Liver.—Hepatic symptoms, even when the liver is displaced to the maximum, are very rare. Pain in the hepatic region is sometimes complained of, but this is probably pleural rather than hepatic. Jaundice, even in a slight degree, is hardly ever observed.

Displacement of the Stomach.—The area of stomach resonance with left side effusions is of very great importance. It is reduced early, even in moderate effusions, and disappears entirely when the effusion becomes large. With a large effusion, I have once or twice been puzzled by finding this region somewhat resonant, but this has occurred chiefly in children, when the stomach has been greatly dilated, and it is altogether unusual. In a case where the diagnosis would lie between pleuritic effusion and pleuritic thickening, or contraction, the presence or absence of the stomach resonance would probably be conclusive.

With a very large effusion, especially of the left side, the œsophagus might be compressed, but if so, the pressure is not sufficient to cause any difficulty in swallowing.

Palpation and Auscultation.—The vocal vibrations, vocal resonance, and breath sounds are, as a rule, entirely absent over the whole side, except, it may be, in two places. One of these is the corresponding interscapular space. Here the lung is collapsed and condensed around the root, and consequently there may be obtained the signs of solid lung; that is to say, the vocal vibrations may be increased, there may be distinct bronchophony, tracheal or bronchial breathing, and sometimes crepitation of a sharp ringing character, such as might be heard over a cavity. The other place is in front, beneath the manubrium sterni. Here it is not at all uncommon to find bronchial or tracheal breathing, and as the percussion is dull, the question might arise whether a mediastinal tumour were not present. However, bronchial or tracheal breathing in this position is very frequent with simple effusion, and disappears when the effusion goes, so that if a mediastinal tumour were present the diagnosis would have to be made by other evidence.

If the lung be adherent at the apex the physical signs may become puzzling. Thus the breath sounds may be bronchial, there may be bronchophony, and occasional crepitation.

It will then be impossible to say that the patient is not suffering from phthisis, and it will be necessary to wait till the fluid has disappeared before this question can be settled. In a very large number of cases all these suspicious signs disappear when the fluid has been removed, and no evidence of apex mischief remains.

Although it is true that, as a rule, the vocal vibrations, vocal resonance, and breath sounds are completely absent over an effusion, still there are exceptional cases in which they are not only present but exaggerated. Besides bronchophony

distinct pectoriloquy may be present, and so may the "signe de sou," *i.e.*, the metallic ring when a coin laid on the chest is tapped by another may be heard clearly transmitted through the fluid.

It is also quite possible to hear as distinct and characteristic bronchial breathing over a pleuritic effusion as is ever heard over a solid lung. This is most frequent in children, but it is far from rare in adults.

Thus I have seen it in a young man with a large serous effusion; the bronchial breathing disappearing when the effusion was withdrawn, appearing again as the effusion reformed, disappearing when it was again removed, and ultimately vanishing entirely as the fluid was absorbed. I remember also the case of a little child of about 3 years of age, in which the diagnosis of a solid tumour was made for this reason. A needle was inserted for the purpose of exploration, but no fluid being obtained, the diagnosis was thought, on that account, to be confirmed, but when the child died the side was found to be filled with fluid and no tumour existed.

The mere knowledge of the fact, in most cases, suffices to prevent error in diagnosis, for the other signs, viz., the character of the dulness and the displacement of organs, make the nature of the disease obvious.

Though the fact is well established, the explanation is difficult.

It might be supposed that the exaggerated breath sounds were merely transmitted by the bony parts of the thorax from those places where they are frequently heard, that is to say, from the inter-scapular space or the upper part of the sternum; but this explanation will certainly not suffice for most cases, because the bronchial breathing may be absent, or much less distinct in the places named, while well marked in the rest of the side. Nor can the explanation be found in irregular adhesion of the lung to the chest walls, for in most cases adhesions are absent, and the lung is separated from the ribs by many inches. Finally, the exaggeration of the breath sounds depends in no way upon the nature of the fluid, for it is quite as frequent with purulent as with serous effusion.

THE CONSEQUENCES OF EFFUSION.

1. Upon the Lung on the affected Side.—When there is a limited effusion at the base, the upper part of the chest in front is often prominent or bulging, and sometimes moves freely. On percussion it is hyper-resonant, sometimes even tympanitic. This is the so-called Skodaic resonance. It is due to relaxation of the pulmonary tissue, *i.e.*, to loss of tone or tension owing to the partial collapse of the lung, and is strictly comparable, I believe, with the similar percussion-note obtained from the lung when removed from the body. A similar condition is met with in the upper part of the chest, with pneumonia of the lower lobe of the lung, the loss of tension in the pulmonary tissue being in this case consequent on impaired nutrition due to the inflammation.

Some authors (*e.g.*, Bristowe and Fagge) adopt a different theory, and refer the hyper-resonance to the diminished vibrating area of the thoracic walls, but I fail to see how this gives any explanation of the fact.

The bruit de pot fêlé, or "cracked-pot sound," is occasionally met with in the hyper-resonant part under these conditions, a fact of interest but of no clinical importance.

The respiratory murmur varies. If the movements are greatly impaired the respiratory murmur is diminished, owing to the defective entry of air. If, however, the movements are free, and still more if they are exaggerated, as they sometimes are, the respiratory murmur may be increased. Then the expiration is prolonged, and both inspiration and expiration are coarse, the respiration being of that character which is described as complementary or puerile, and not always easy to distinguish from bronchial breathing.

Well-marked bronchial or even amphoric breathing is by no means rare at the apex above a large effusion without any serious change in the lung to account for it. With a large effusion where the apex has been dull I have seen it appear as the fluid has been drawn off, disappear again as the fluid re-accumulated, and appear once more when the fluid was again withdrawn.

The vascular congestion of the upper parts of the lung may betray itself by no physical signs, but if it does the signs are those of bronchitis, viz., rhonchus, sibilus, and possibly some crepitation. Under these circumstances it may not be easy to say what the exact condition of the apex may be, for the physical signs may raise the suspicion of phthisis, a suspicion which can be only allayed by the course the case runs, and by the disappearance of these physical signs when the effusion has been removed.

2. **Upon the Lung of the opposite Side.**—The collapse of the opposite lung must be considerable when it is considered that the diaphragm on that side remains at its usual level, whilst the capacity of the side is reduced by the space occupied by the displaced organs. With one lung completely and the other considerably collapsed, the respiratory capacity must be reduced to not much more than a third of the normal. To make good the respiratory deficiency the movements are increased in rapidity and in extent, and in consequence the breath sounds become puerile or complementary.

So long as the lung, thus reduced in capacity, is able to meet the demands made upon it, nothing but puerile breathing will be heard, but as soon as the work proves too much for it, failure shows itself in the physical signs of congestion, *i.e.*, of bronchitis, namely, rhonchus, sibilus and crepitation. These signs, showing that the lungs are overworked and beginning to give way, are of grave omen, for unless the congestion is relieved, *i.e.*, unless paracentesis is quickly performed, the bronchitis will rapidly increase and end in suffocation. The development, therefore, of the signs of bronchitis in the opposite lung becomes an indication for immediate paracentesis.

THE SYMPTOMS OF EFFUSION.—The symptoms are often much less than the physical signs would lead us to expect. Thus one side of the chest may be almost full of fluid without producing any more symptoms than perhaps a little shortness of breath on exertion. On the other hand, the symptoms may be well marked when the effusion is moderate, and they are often more marked in previously healthy, full-blooded persons than in the anæmic or feeble. The reasons of this lie, first in the rate at which the fluid has formed, and secondly in the effect produced on the other lung; for the symptoms depend not only upon the compression of the one lung by the fluid, but upon the congestion of the other, and the consequent embarrassment of the circulation in it. So it is that a small effusion, developing rapidly, will often produce more acute symptoms than a large one which has developed slowly.

The chief symptom is, of course, *dyspnœa*; but even without marked dyspnœa the respirations are often much accelerated.

With a moderate effusion this may depend, as in dry pleurisy, upon the pain and the consequent shallow breathing, but if the temperature be high the fever also must be responsible in part for it. When the effusion is large the dyspnœa depends chiefly upon the size of the effusion and the consequent congestion of the opposite lung.

It is obvious that with one lung thrown more or less completely out of work very slight changes in the other may be attended with a great exacerbation of symptoms, and that with a large effusion dyspnœa may develop suddenly and rapidly become urgent.

As stated, this is generally associated with the signs of congestion, *i.e.*, of bronchitis on the opposite side; but the same may occur with the onset of certain complications, of which pericarditis is the most important. If the signs of congestion of the opposite lung be present, even if there be not much dyspnœa at the time, it will be well to take them as an indication for the removal of the fluid at once, and not to wait until the dyspnœa becomes pronounced, for it may then be too late to give permanent relief.

Cyanosis stands in direct relation with the amount of dyspnœa, and though in many cases the complexion may be a little dusky, anything deserving the name of cyanosis is uncommon.

Nowadays, except where large effusions have formed with very great rapidity, extreme dyspnœa and cyanosis are rarely met with, for in most cases the diagnosis of effusion is made early, and the fluid removed.

Pain is by no means so conspicuous a symptom of pleuritic effusion as it is of dry pleurisy, and it is often completely absent. With a localised effusion the pleuritic stitch may be felt at the margin where the two layers of the pleura are in contact; and when an effusion has been absorbed and the pleural surfaces come once more into apposition, the pain may return and even friction be again heard (*Redux Pain and Friction*).

Even when an empysema is pointing, the pain felt is not so much the characteristic sharp stitch of pleurisy as the throbbing ache of an abscess, and the part is tender to the touch. With very large effusions the pain has the character of distension, of stretching, rather than of stitch, but the feeling of distension may be distressing.

Cough is as a rule absent, or unattended with expectoration. It is more common with small effusions than with large ones, and in the latter often depends upon bronchitis of the opposite lung, but even in some cases of serous effusions severe paroxysms of coughing may occur without any very obvious reason.

When an empyema is on the point of bursting through the lung, a frequent, violent, paroxysmal cough may set in, and then become an indication for immediate paracentesis. If the empyema has burst, and is discharging freely through the lung, the cough, though frequent, is often short and easy, but it varies greatly under different circumstances; thus it may be greatly aggravated by position; occur in periodical paroxysms of very great severity, and be accompanied with a profuse discharge of matter, just as happens with large cavities in the lung, while in the intervals between the paroxysms there may be no cough to speak of at all.

Fever.—The temperature varies according to the stage of the disease in which the patient is seen, and if the acute stage be passed the temperature may be normal whether the effusion be serous or purulent. In either case it is always raised at the commencement, as it is in dry pleurisy. The temperature in the later stages of pleuritic effusion varies a good deal according to the cause of the disease, the course of the case, and the nature of the effusion. As a rule, a well-marked hectic temperature with wide daily oscillations, though not conclusive, is strongly suggestive of empyema, especially if the physical signs point to only a small collection of fluid.

But in all cases it must be remembered that the fever may be due, not so much to the effusion as to the disease which has produced the effusion, for instance, tubercle.

Decubitus.—The patient naturally takes that position in which he breathes easiest and suffers least pain; thus, in the early stage of acute pleurisy, as a rule, he lies on the sound side, because pressure on the other increases pain. When fluid forms the pain is relieved, and the patient turns on to the affected side, in order to leave the opposite lung freer to move.

With an effusion of moderate size the patients usually prefer to lie in an oblique position with the shoulder slightly raised, and the body turned slightly to the affected side. In this position the weight of the fluid is largely supported by the lower ribs, and thus the upper part of the affected lung is able to take its part in breathing.

With a large effusion the patients prefer to lie almost flat upon the back with the head but little raised, or else turned over completely to the side on which the fluid is. In this position they may not suffer much difficulty in breathing. But dyspnœa becomes considerable if they sit up, because the fluid is then thrown upon the diaphragm; or if they turn over on to the opposite side, because then not only are the movements of that side checked, but the weight of the fluid is thrown upon that lung and its respiratory capacity still further diminished.

So far the account given of the physical signs and effects of an effusion applies equally to all forms of effusion, sero-fibrinous or purulent. From this point it will be necessary to deal with these two forms separately.

THE DIAGNOSIS BETWEEN SEROUS AND PURULENT EFFUSIONS.—The diagnosis between serous and purulent effusions is often impossible without the aid of an exploring needle, unless it happens that the signs of pointing are present. The chances are in favour of empyema if the patient be a child, if the fever be long-continued and of a markedly hectic type, if there be repeated chills and much sleep-sweating, if the patient looks pale and ill and has lost much flesh, and if the signs of pleurisy develop in the course of some septic disease or specific fever. If all these conditions are combined, the diagnosis is very probable, but not one of them is conclusive in itself; thus, children often have serous effusion; the temperature may be markedly hectic with a serous effusion, while there may be a normal or even subnormal temperature with empyema, and in either case the hectic fever may be due not to the effusion but to the disease that caused it, *e.g.*, to tubercle.

Even with serous fluid there are sometimes repeated shiverings and much sweating, while the temperature may be hectic and remain high for long periods of time.

Very rapid formation of fluid is strongly in favour of serous effusion as opposed to empyema, for pus accumulates slowly, and to reach the same size an empyema will take as many days as a serous effusion may hours.

It has been said that the duration of a pleuritic effusion is some guide as to its nature, and that if the signs continue more than a few weeks (some authors actually specify a shorter date) the probabilities are strongly in favour of its proving to be pus. These statements date from the time when the needle was not used for exploratory purposes, and they amount to no more than this, that in a case in which the effusion was thought at first to be serous, it proved after a time to be purulent. In the days when antiseptic precautions were not strictly observed, it was of course common enough to see an effusion which, when first tapped was serous, on later tappings prove to be purulent. The real explanation of this is to be found in the infection of the serous effusion as the result of the operation. There is no proof that a serous effusion untouched is likely to become purulent by mere lapse of time. Indeed, experience proves the contrary, and shows that a serous effusion may remain serous for months or even years, that once serous the effusion remains serous, unless infected with pyogenic organisms, while in most instances purulent effusions are purulent from the commencement, as may be demonstrated by exploration with the needle.

There are some undoubted instances in which serous effusion has spontaneously changed its character and become sero-purulent or purulent (as, indeed, there are also rare instances of the contrary), but they are few and far between,

and the general conclusion may be safely drawn, that if a serous effusion become purulent it is due to fresh infection with pyogenic organisms, and that these organisms have been introduced either from without by dirty instruments, or from within from some infective focus seated usually in the lung.

Dry Tapping.—In all doubtful cases of diagnosis it is well to explore with a needle. If fluid is found, well and good; if not, the result is called "a dry tap." It would be better called a failure, for it does not necessarily follow that there is no fluid there.

1. In the first place, we must be satisfied that the fault does not lie in the apparatus, as it frequently does; that is, we must see that the syringe or aspirator, as the case may be, works properly, and that the needle is clear.

This, of course, should be ascertained as a matter of routine before the needle is used.

The needle may become plugged after it has passed through the thorax, and to settle this it may be well to pass the blunt probe through it. This cause of a dry tap is, however, quite uncommon, for although the needle may become plugged during paracentesis, so that little fluid can be drawn off, it hardly ever happens that enough is not obtained to serve the purposes of diagnosis.

2. The more common cause of failure is that the needle is not itself in the pleural cavity; *e.g.* (*a*) the pleura may be much thickened, and thus the needle be not thrust far enough to pierce it; or (*b*) the effusion may be quite superficial, and the needle have been thrust in too far, and this is not unlikely to happen if the lung be solid, as with effusion occurring in the course of pneumonia; or (*c*) the effusion may have been entirely missed.

Generally it is quite easy to tell by the touch whether the needle is in a cavity or not, for in a cavity the point is felt to be free when the needle is moved from side to side, whereas in solid tissue it is held fast.

3. Lastly, there may be some unusual form of pleurisy present, *e.g.*, the fluid or exudation may be gelatinous, and, as in the areolar form, held in a meshwork formed of adhesions.

There is one theoretical condition which has been suggested to explain a dry tap, viz., that the walls of the pleural cavity are extremely rigid. Of course when fluid is withdrawn from the pleura, the side contracts or the lung expands to take the place of the fluid withdrawn. If, then, the lungs were so tightly bound down and the walls so rigid that they would not yield, theoretically no fluid could be obtained. As a matter of fact these conditions are impossible, and although in some rare cases the parts might be unyielding enough to prevent more than a fraction of the fluid being taken away, still a certain amount can always be withdrawn, and if the needle be clear and has reached the cavity, whatever the nature of the fluid may be, enough is obtained for the purposes of diagnosis.

If the tapping be dry, while the physical signs point clearly to the existence of fluid, it will be safest to draw the conclusion that the needle has not reached the cavity and to repeat the operation in another place or in another way.

Simple as exploratory puncture or paracentesis for pleuritic effusion usually is, it is not always as easy as it seems, and it may be extremely difficult. Unfortunately there are no definite means of knowing when the case is likely to be simple and when difficult, so that it is always well to be prepared for unexpected difficulties. This, however, holds good more in empyema than in ordinary serous effusion.

It remains to speak of a curious result occasionally observed after tapping. It may happen after a dry tap, or a puncture which has removed only a few drachms of fluid, that great relief has been experienced by the patient, and that, as the apparent result of the puncture, the physical signs have begun to improve, and rapid resolution has occurred. The amount removed is too small to have had any effect upon the mechanical conditions in the pleura, and what explanation is to be given of this extraordinary occurrence I have no idea; but it is observed after puncture, not of the pleura only, but of other parts of the body; too often it would seem for its occurrence to be regarded as a mere coincidence.

62. SERO-FIBRINOUS EFFUSION.

PLEURITIS EXUDATIVA SERO-FIBRINOSA.

It is impossible, at the commencement of a case of acute pleuritis, to predict whether there will be effusion or not.

The onset of the disease is the same in either case, with a stitch in the side and friction; but as fluid forms, the pain and friction diminish or disappear. In at least three cases of acute pleuritis out of four the affection begins with acute

symptoms, but in some of the remainder the symptoms are indefinite, and in a few, fluid is found without anything in the history to indicate its time or mode of onset.

A rigor marking the onset is rare, so is frontal headache. Sickness and vomiting are less uncommon and sometimes are excessive; in one case they lasted a week before any other definite signs developed, except fever.

Occasionally the pains are general, and felt all over the body and in the limbs, just as in the onset of specific fever.

The initial dry stage may last a few hours only or continue for some days.

The formation of fluid, once begun, is usually continuously progressive, but its increase is sometimes irregular, the fluid reaching a certain point, remaining stationary for a time or even seeming to diminish, and then increasing again.

When the fluid has ceased to increase, it usually remains stationary for a time, and then begins to disappear, slowly at first, and afterwards with increasing rapidity.

The rate of effusion varies greatly, but in most cases it is not very rapid; half a pint a day would be above the average. It will thus require several days, or even two or three weeks, for the fluid to reach any large amount. On the other hand, the rapidity with which effusions may sometimes develop is surprising.

I have seen the side filled with fluid to the maximum in less than three days from the commencement of illness, so as to require paracentesis without delay. Eighty ounces were removed, but the operation had to be repeated within three days because the chest seemed as full as ever.

Feltz[1] records a case in which 105 ounces accumulated in three days, *i.e.*, the fluid was formed at the rate of an ounce and a half per hour.

Fortunately these cases are rare, for the symptoms they produce are very severe, and the risk to life considerable, unless paracentesis is performed soon, and repeated as frequently as may be necessary.

Large effusions never disappear at a similar rate, though small amounts of fluid may come and go with great rapidity.

When the pleurisy is associated with consolidation of the lung, as where it follows pneumonia, a small amount of effusion may cause very extensive dulness, which may form rapidly and disappear as rapidly.

In a patient in a late stage of pneumonia I have seen dulness develop over the whole side in twenty-four hours, and in another twenty-four hours entirely disappear; the explanation, no doubt, being that in this case a large amount of the lung was consolidated, so that a little fluid spread itself out in a thin layer over a very wide surface.

The **temperature** varies a good deal, and though at the time that the case comes under observation—some days or weeks, it may be, after the onset of the disease—the temperature may be low, still in the initial stage it is almost invariably raised. (*Cf.* Charts I. and II., p. 671.)

In acute cases it may rise to 103°, but rarely beyond, and that only for the first few days. It more commonly ranges about 102°. It is remittent in type, somewhat hectic in character, and frequently with wide daily oscillations. In this respect it is very like that which is met with in empyema, and, as has been stated, the temperature is but a poor indication as to the nature of the effusion.

Usually the fever continues for two or three weeks; but it may last longer, even six or seven weeks, and that in a case which appears simple, and ends in complete recovery.

The fever is usually little, if at all, affected by paracentesis; and though, now and then, tapping may be followed by a sudden fall to the normal, this is much rarer in serous effusion than it is in empyema.

[1] *Gaz. d. Hôp.*, 1870, p. 249.

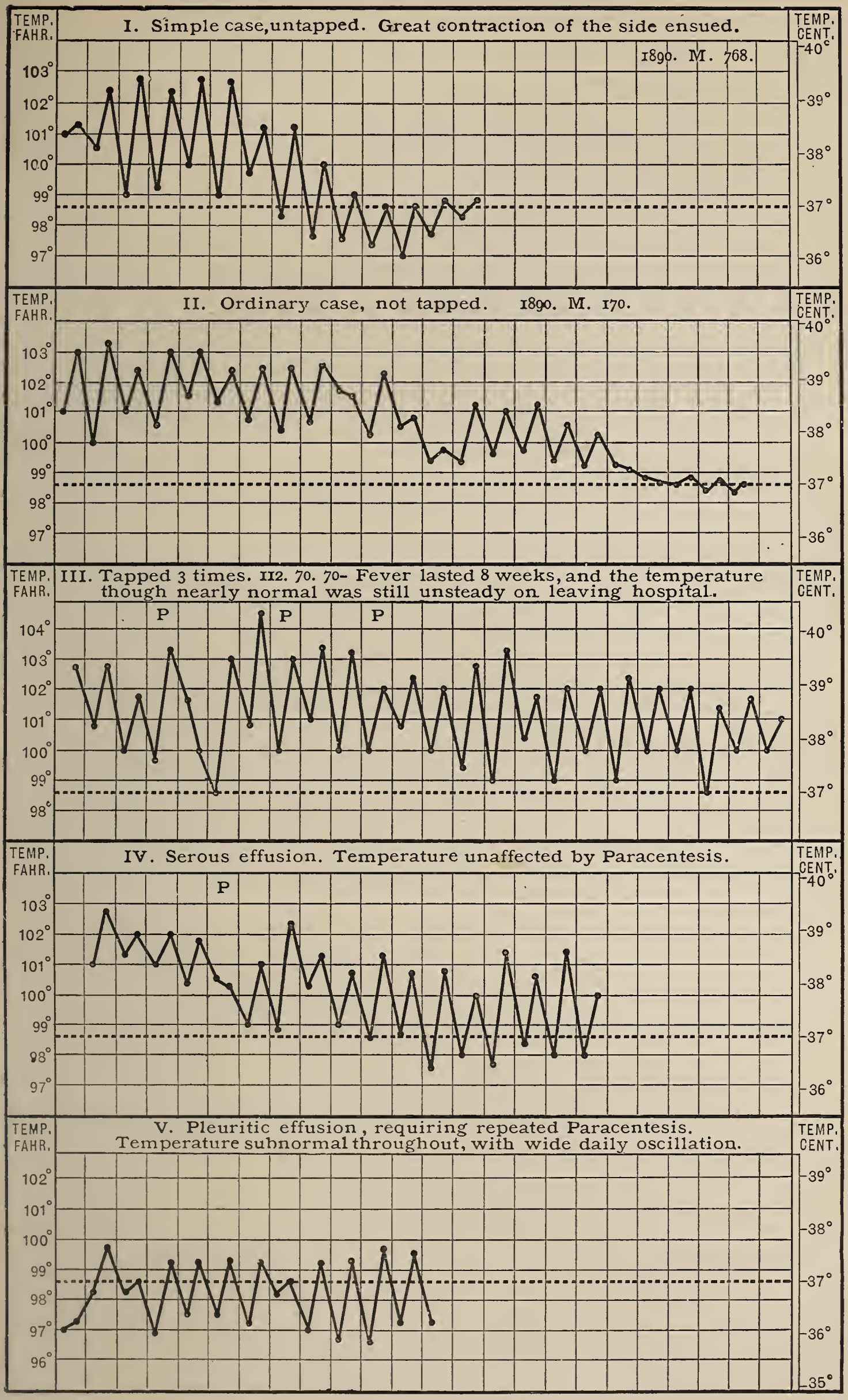

I. Simple case, untapped. Great contraction of the side ensued.
1890. M. 768.
II. Ordinary case, not tapped. 1890. M. 170.
III. Tapped 3 times. 112. 70. 70- Fever lasted 8 weeks, and the temperature though nearly normal was still unsteady on leaving hospital.
P
IV. Serous effusion. Temperature unaffected by Paracentesis.
V. Pleuritic effusion, requiring repeated Paracentesis. Temperature subnormal throughout, with wide daily oscillation.
TEMP. FAHR.
TEMP. CENT.

Fig. 149.

The oscillations may be considerable, even when the maximum temperature is comparatively low, and may be even continued into convalescence, though the maximum be never above 99°, or even exceed the normal.

Occasionally the fever recrudesces, and that too without fresh effusion or other obvious cause; though, of course, with a fresh effusion a rise of temperature is likely to occur.

The fever is sometimes very prolonged, and continues for some time after the effusion has in great part disappeared. The explanation lies, no doubt, in the presence of tubercle. In other words, phthisis may commence as a pleurisy with or without effusion.

The earliest evidence that a pleuritic effusion is ceasing to increase is usually given by the fall of temperature, with which there is often associated a fall in the pulse- and respiration-rate, and improvement in the general condition.

The temperature usually falls gradually, and several days may elapse before it reaches the normal.

Pleuritic effusion sometimes terminates by crisis, with sweating or diuresis, as pneumonia does, but this is quite unusual.

Soon the physical signs show that the fluid is being absorbed; the percussion resonance improves, first at the upper part of the chest in front; the line of absolute dulness descends; and the organs begin to return to their normal places.

Over the seat of fluid, even before the percussion note has appreciably altered, the vocal resonance may be heard; the vocal vibrations return later; and, last of all, the breath-sounds become audible; but the percussion note may remain impaired long after all other signs of fluid have disappeared.

Contraction of the Side.—As the fluid is absorbed the side becomes somewhat contracted, and the movements continue for some time impaired.

Theoretically it is, of course, possible that as the fluid is absorbed the lung may re-expand and no flattening be produced, but, as a matter of fact, even in the slightest cases, this rarely happens, probably for the reason that the lung does not all at once recover its normal elasticity or the intercostal muscles resume their normal action.

When the two layers of the pleura come again into contact, pain and friction may return (Redux friction), and continue until adhesion has taken place, and then disappear; but friction may continue a long time.

Thus in one case it was still audible and apparently unchanged nine months after the patient had recovered from a pleuritic effusion, for which paracentesis had been necessary.

Even without any actual return of pleurisy, patients may have attacks of pain in the seat of lesion from time to time, if they get out of health or catch cold, just as an old sprain or damaged joint may, under similar circumstances, give trouble.

Apparently adhesion between the two layers of the pleura does not always take place, for occasionally after the lapse of time an effusion develops again on the same side as before.

In one case, a girl of 10, the first pleuritic effusion occurred seven years previously, and in a man of 30, seven years before.

The same thing is seen with empyema also.

Thus a man of 48 was operated on for empyema and cured; five months later he developed a small serous effusion on the same side.

The flattening is, in most cases, brought about by the adhesion which has taken place between the two layers of the pleura, and by the contraction of the connective tissue which has been produced as the result of the pleurisy.

The contraction takes place in three directions.

1. Between the side of the thorax and the root of the lung or the spinal column ; this happens when the two layers of the pleura are adherent, and the interlobar or main septa of the lung also involved.
2. Round the lung itself, when an extensive surface of the pulmonary pleura is involved, the lung being thus compressed and made smaller.
3. Between the ribs themselves, so that they are drawn towards one another. This happens when the costal pleura is widely affected.

The contraction of the side is, of course, most marked when these three factors combine, and then considerable deformity may be produced.

When the two layers of the pleura are simply adherent there may be little or no deformity. It is not uncommon to find the pleura obliterated over a large part, or even the whole of one lung, without anything whatever in the physical signs to give evidence of it. In most of these cases the lung is healthy and retains its elasticity.

If the lung itself or its surface be also fibrotic, contraction must necessarily occur and be permanent. The most obvious examples of this are seen with phthisis at the apex or with interstitial pneumonia at the base.

Even in extreme cases the amount of external deformity will depend upon the degree to which the mediastinum is still free to move, or has been fixed by the pleurisy.

The inflammation often spreads from the pleura to the tissues of the mediastinum, and then the mediastinum may be fixed permanently in any position which it happens at the time to occupy.

When the heart has been displaced by the effusion far into the opposite side, and becomes fixed there, the contraction of the affected side will be considerable, and there will be great impairment of respiratory power, because, though the opposite lung may still remain healthy, it is, to a great extent, permanently collapsed.

If the mediastinum be not fixed, but be free to move, it may be drawn, together with the organs it contains, farther and farther over into the affected side as the contraction occurs, and the opposite lung, if it be healthy, may undergo compensatory hypertrophy. Ultimately these two processes, viz., the displacement of the mediastinum and the complementary hypertrophy of the lung, may completely compensate for the contraction that has taken place, so that no external deformity whatever of the thorax may be evident, and sometimes even no defect of respiratory movement on the affected side. The deformity which the contraction of the pleura has then produced is internal and not external.

I have seen, in an adult, the heart so far displaced to the right side that the case was regarded as one of transposition of viscera, but it was obvious from the history and other physical signs that the displacement of the heart was really due to a pleurisy which had occurred during childhood.

If, however, the mediastinum benot free, the effects of pleuritic contraction will show themselves upon the ribs, and the side will be greatly drawn in, and the movements, of course, correspondingly impaired. In such a case the deformity may be considerable, greatest, of course, in those cases where the ribs are

soft and yielding; in children, therefore, or, in some cases, in young adults. Still, even then, the amount of deformity following serous effusion rarely reaches anything like that extreme degree which is common after empyema.

I have seen the superficial veins become greatly dilated over the whole side, lower as well as upper parts, when there has been considerable flattening. This, no doubt, indicated much fibrotic thickening of the parietal pleura and consequent obstruction of the intercostal veins. This, which is so commonly seen at the apex in chronic tubercular pleurisy, is distinctly rare in other parts of the chest, and especially at the base and lower parts of the axilla.

It is very remarkable how the deformities produced by pleurisy may rectify themselves in the course of time.

No doubt this is brought about in a similar way to that in which a stiff joint in time recovers its movements. Perhaps the adhesions which have formed may actually resolve and disappear, but, although this is conceivable, it is difficult to prove in any case, and certainly does not often occur. Sometimes adhesions snap in the pleura as they do in a joint, the patient being conscious of this, and developing for a few days the signs of dry pleurisy in the spot where the pain is felt.

In extreme cases, where the deformity rectifies itself this result is brought about, as already stated, by the change in the position of the mediastinum, and by the compensatory hypertrophy of the opposite lung. Even when the pleurisy is partial the compensatory hypertrophy of the rest of the affected lung may be quite sufficient in time entirely to mask its effects, the essential condition being that the lung itself should be healthy, for, if it be not, compensatory hypertrophy does not occur.

One other condition still remains to be considered, viz., that in which, after the lung has come out as far as it can and the ribs have fallen in, still a space is left between the two surfaces of the pleura. The space must be occupied by something, and it will remain permanently filled with fluid. This is the condition to which the term *hydrops ex vacuo* is applied.

The conditions just described as the result of pleuritis exudativa, viz., pleural adhesion, pleural thickening, pleural contraction, and persistent effusion are often embraced by the one term *pleuritis chronica.* The term 'chronic' in this, as in other cases, is not a good one, for it is applied at one time to the pathological condition and at another to the clinical duration of the case. Strictly speaking, the term "pleuritis chronica" should be applied only to cases of progressive disease of the pleura of inflammatory origin, which are not and never have been acute, such pleurisy as is so frequently met with in phthisis. An acute pleurisy may last a long time, and may, on that account, be called chronic, and a pleurisy of long duration may be found associated with lesions which are not chronic in the pathological sense. Clinically the term is often applied to cases in a purely arbitrary fashion: thus, it is stated sometimes that if a pleuritic effusion has lasted more than a month it may be called "chronic"; yet, though lasting a long time, it may never cease to be acute, and may in the end resolve completely. The pathological conditions which result from pleuritis do not differ from those which are the result of non-inflammatory causes; so that the term "pleuritis chronica" is both pathologically and clinically confusing.

DURATION.—The duration of pleuritic effusion is very variable. Thus in a slight case, where the effusion is small, it may take a week or ten days to develop, and probably as long for the fluid to disappear, so that the average

duration of a slight case will not be less than two to three weeks. The larger the effusion, the longer will, as a rule, be the duration of the case.

The rate of absorption varies as much as the rate of effusion, and it is this which largely determines the duration of the case.

Where the effusion is a very large one there are mechanical difficulties in the way of its absorption, as has been already explained, and such an effusion may continue for months unchanged, although even then, if paracentesis be performed, and some of the fluid removed, the rest may disappear and the case rapidly clear up.

Lichtheim[1] records an instance of the spontaneous disappearance of an effusion which had lasted for six months, and Wilson Fox[2] mentions cases of the cure by a simple paracentesis of an effusion which had lasted 5½, 6 and 8 months respectively.

In cases of large effusion of long duration, however, such a favourable result after paracentesis is by no means of common occurrence. What generally happens is that the fluid re-forms and paracentesis has to be repeated, it may be, many times, and recovery is gradual and slow.

The most striking case I shall refer to later, in which a young lady, known to have fluid in her right side, was allowed to go on without interference for eighteen months, and in spite of that, paracentesis showed that the lung was still capable of complete re-expansion.

MORTALITY AND CAUSE OF DEATH.—The mortality of acute pleurisy, whether dry or with effusion, is small. The total mortality in ten years at St. Bartholomew's Hospital works out at 7 per cent., and it is interesting to observe that the mortality of the cases of pleuritis sicca is rather higher than that with serous effusion, but it must be remembered that the mortality is very much less than this really, because it is only the severe cases that are admitted into the hospital.

Thus Ewald gives a mortality of only 2·7 per cent. in a series of 178 cases.

In most cases when death occurs, it is due not so much to the effusion itself as to the disease of which the pleuritic effusion has been a complication, for instance tubercle; or to some complication of the pleurisy, *e.g.*, pericarditis; or to some affection such as bronchitis, pneumonia, or pleurisy, on the other side of the chest, for with one lung completely collapsed and useless, a very slight complication in the other may cause urgent symptoms, and bring the case to an end.

Mode of Death.—In most of the fatal cases the patients die slowly with the signs of asphyxia. This is associated with the signs of pulmonary congestion or bronchitis in the opposite lung.

As already stated, the evidence that the opposite lung is failing from the stress thrown upon it, is given by the signs of bronchitis. They are at first slight and of gradual onset, but sometimes develop unexpectedly, with great rapidity and intensity. However slight they may be, they are an indication for immediate paracentesis. If paracentesis be performed at once, the congestion is relieved and the signs disappear; but if paracentesis be delayed, the lungs may not recover themselves after the removal of the fluid, and the patients die with gradually increasing cyanosis and suffocation, much as they do with bronchitis.

Sudden Death in Pleural Effusion.—It sometimes happens that death occurs suddenly and unexpectedly. This is an extremely rare event now that paracentesis is performed early and repeated as often as may be necessary. All

[1] *Volkmann's Sammlung.* [2] Wilson Fox, *l.c.*, 1014.

TABLE showing Age, Distribution, Sex, and Mortality for Dry Pleurisy and Pleurisy with Serous Effusion, for 10 years (1883 to 1892 inclusive).

(Taken from the Statistical Tables of St. Bartholomew's Hospital.)

Disease.	Total.	Dischd.		Died.		Under 5.				—10.				—15.				—20.				—30.				—40.				—50.				—60.				Over 60.			
		M.	F.	M.	F.	Dischd. M.	Dischd. F.	Died. M.	Died. F.	Dischd. M.	Dischd. F.	Died. M.	Died. F.	Dischd. M.	Dischd. F.	Died. M.	Died. F.	Dischd. M.	Dischd. F.	Died. M.	Died. F.	Dischd. M.	Dischd. F.	Died. M.	Died. F.	Dischd. M.	Dischd. F.	Died. M.	Died. F.	Dischd. M.	Dischd. F.	Died. M.	Died. F.	Dischd. M.	Dischd. F.	Died. M.	Died. F.	Dischd. M.	Dischd. F.	Died. M.	Died. F.
Total cases,	638	423	170	33	12	10	2	6	4	35	20	3	...	27	15	...	1	35	20	1	...	120	54	3	2	102	35	5	1	65	15	4	2	22	6	7	1	6	3	4	1
Per-centage,	100	66	27	5	2	3·4				9·0				6·8				8·8				28				22·4				13·5				5·7				2·5			
																						50·4																			
Dry Pleurisy,[1]	238	149	73	14	4	Giving mortality of 7·6 per cent.																																					
Pleurisy with Effusion,	395	271	97	17	8	,,		,,		6·3		,,																													

Combined, nearly 7·0 per cent.

Relative Mortality of Pleurisy with Effusion at the different Age Periods.

Total Average,	6·3 per cent.	45·5	5·2	2·3	1·8	2·8	4·6	7·0	22·0	35·6

The table also shows how greatly the mortality differs at different periods of life. Thus, in the middle period of life, when the number of cases of pleuritic effusion is the largest, the mortality is at its lowest, and rises rapidly towards the end of life, when pleuritic effusion is a far less frequent affection. The tables also seem to show that the mortality is at its highest in the early years of life, that is to say, before the age of 5, but the numbers are too small to be conclusive, and I should not have thought they really represented the facts, judging by my own clinical observation.

[1] Five cases were not adequately described.

the cases of sudden death described in pleuritic effusion do not equally deserve the name, for in some the death would be better described as rapid rather than sudden, as it may take some hours to occur instead of a few minutes.

Sometimes patients have been found dead in bed without anything having occurred during the night to draw the nurse's attention to them, or, while sitting up in bed, they have fallen back upon the pillow, given a few gasps, and expired. These are usually regarded as cases of cardiac syncope, which they closely resemble.

In other cases the symptoms are more like those of pulmonary embolism, for the patient may without cause be suddenly seized with intense dyspnœa, and become rapidly cyanosed, with a distended right heart and a rapidly falling pulse, being at first conscious and in the greatest agony of mind and body, but soon relieved from suffering by unconsciousness, and dying of asphyxia, sometimes in a few minutes, at others after a long struggle, lasting, it may be, for several hours.

Among my own cases I have seen three cases of sudden death in the course of pleuritic effusion.

In one, acute pneumonia developed after the pleurisy, and this should hardly count.

In another, a man 46 years of age had been ill a fortnight; the chest was tapped, and 20 ounces of serum removed with some difficulty. The next day he was seized with greatly aggravated dyspnœa, and brought up a large, tough, whitish cast of a large bronchus. This did not materially relieve the dyspnœa, except for a very short time, and he died a few hours later. The temperature and history did not suggest pneumonia, and there was no previous evidence of plastic bronchitis, nor anything to suggest diphtheria. No *post-mortem* could be obtained, so that the exact diagnosis remained uncertain.

In the third case, a man, aged 35, was admitted with a large effusion, which improved rapidly, so that in ten days he was allowed to get up. This was immediately followed by a relapse, the temperature rose to 103°, and there was much dyspnœa. Paracentesis was performed; 98 ounces of serous fluid removed and great relief given. Thirty-six hours later, without obvious cause, dyspnœa became urgent, and in a few hours the patient died. On *post-mortem* examination no cause could be found, except that the heart was pale and flabby. There was no clot in any of the vessels, nor anything in the lung to account for death.

Rare as sudden death in pleural effusion is at any age, the liability to it is greater with advancing years. It is very rare in children, although some few instances are recorded.

It seems to be more frequent in pleurisies of recent date than in those of longer duration, and Lebert states that the risk of this event is less the longer the effusion has lasted.

Various explanations have been offered.

1. Some authors have attributed it to mechanical interference with the circulation through the large vessels consequent on the displacement of the heart. Thus Chomel and Trousseau referred it to twisting or stretching of the large vessels at the base of the heart; while Bartels and others referred it to stretching or kinking of the vena cava inferior; the result being due to defective blood supply, in the former case to the brain, in the latter to the heart itself. *Post-mortem* evidence is lacking in confirmation of either of these theories; for the twisting of the vessels cannot be demonstrated on *post-mortem* examination, nor can it be produced experimentally.

In either case these changes, if they occur, ought to be greatest when the effusion is large and left-sided; yet cases of sudden death occur with equal frequency with right-sided effusion as with left, and in a large number of cases the effusion has been only of moderate size. Thus, of Dieulafoy's 40 cases, in two-thirds the effusion was on the right side, while in a large number the amount of fluid did not exceed 60 to 70 ounces.

Wilson Fox's results are much the same, except that the proportion of right- to left-sided effusion was equal.

2. Another mechanical explanation is that of Garland, in which it is supposed that the heart itself is pressed upon by the fluid, especially the right auricle. If this were so, instances of sudden death ought to be more common when the effusion is on the right side and of considerable dimen-

sions, but this is not the case, nor is sudden death a common occurrence in cases where the heart is obviously greatly compressed, as with new-growth in the mediastinum or with pericardial effusion.

3. A more satisfactory explanation is that suggested by Raynaud, See, and others, namely, that the sudden death is due to cardiac syncope, consequent on myocardial degeneration, the result, it may be, of some complication like pericarditis or some general failure of nutrition, and this theory acquires some general support by the increasing frequency of the event as age advances.

4. The last, and by far the most satisfactory, explanation is that which refers the death to cardiac clotting or to pulmonary embolism.

The two modes of death, as already stated, are syncope and asphyxia. Many of the cases of syncope are undoubtedly cardiac in origin, while the cases of asphyxia are best explained by clotting and embolism.

Clinically the symptoms of the asphyxia group are exactly those of pulmonary embolism.

In some of the cases *ante-mortem* clots are found in the heart, extending sometimes continuously a long distance into the branches of the pulmonary artery; but so they often are in other cases to which no sudden symptoms have occurred; hence it is not the clotting itself which is the cause of death. More probably it is the detachment of this clot in whole or in part, with the consequent embolism of the pulmonary artery, that explains the sudden onset of symptoms. Such clots have been actually found in these and similar cases lying coiled up in the pulmonary artery, and when uncoiled they could be put back into their original place of formation in the heart, thus distinctly showing their origin.

It is these last two views, viz., cardiac syncope and pulmonary embolism, that seem to me to explain most satisfactorily both the pathological and clinical conditions.

COMPLICATIONS.—Most of the so-called complications of serous pleurisy, like congestion of the opposite lung, hardly deserve the name, for they are really the consequences of the large effusion left long unrelieved. There are, however, three affections which may arise as real complications of the disease, viz., pneumonia, bronchitis, and pericarditis, all of them rare. Independent *bronchitis* is, perhaps, the rarest of all. *Pneumonia* is next rarest, for although pleurisy is a very common result of pneumonia, still the converse is rare.

Pericarditis, resulting from the spreading of the inflammation from the pleura to the pericardium, is not altogether rare, though here again it is more common to meet with pleurisy as the result of pericarditis than the contrary. When pericarditis occurs, it is usually of the dry kind, does not produce any grave symptoms, and usually resolves completely. It may, however, be a grave complication, and is now and then the cause of death.

Clotting in the heart, with its results, *e.g.*, the detachment of a portion and its lodgment in the pulmonary artery or the vessels of the brain, has been already referred to when speaking of the causes and modes of death. This would be better described as an accident rather than a complication.

PROGNOSIS.—The *immediate* prognosis of serous effusion is favourable in the absence of any serious and unusual complication. The only danger that is likely to arise is in connection with the size of the effusion, and that can be obviated by paracentesis.

The *ultimate* prognosis depends upon the cause of the disease, in other words, the prognosis is that of the original disease of which the pleurisy is a consequence. It is in the prognosis of the cases of apparently primary pleurisy that the interest centres. It has been shown that at least 50 per cent. of these are probably of tubercular origin; that being so, the ultimate prognosis is, of course, doubtful; yet a very large number of them do well, and symptoms of tubercle elsewhere do not develop. Indeed, it seems as if we might draw the conclusion that tubercle of the pleura, like tubercle of the peritoneum, is one of the more curable forms of tuberculosis; or to express it differently, that patients with tubercle of the pleura, as with tubercle of the peritoneum, may get apparently well of their disease, and remain well for a very long time without any other signs of tubercle developing.

DIAGNOSIS.—The diagnosis of serous effusion is, in most cases, easy, but there are certain conditions of the lung, of the pericardium, of the mediastinum, of the walls of the chest, and finally of the parts below the diaphragm, in which the exact diagnosis may present very great difficulties.

Certain Conditions of the Lung.—Pneumonia.—The physical signs of pneumonia and pleuritic effusion are, as a rule, so characteristic and so opposed to one another that no confusion between them can arise; yet sometimes the physical signs are very misleading, and may cause the one to be mistaken for the other; thus, in pneumonia the vocal vibrations, vocal resonance, and breath sounds, instead of being greatly exaggerated, may be entirely absent, just as they are with effusion. The explanation lies in the fact that the tubes are plugged with secretion. As soon, however, as the secretion is removed and the tubes become pervious again, the characteristic signs of pneumonia appear and the difficulties vanish.

In the same way, when the air-tubes have been long obstructed by a foreign body, or by new-growth, the part of the lung corresponding with the obstructed air-tubes passes into a condition of *solid œdema*, and over this the voice- and breath-sounds are absent. As in many of these cases the temperature rises in consequence of inflammatory or degenerative changes in the collapsed lung, the diagnosis may become very difficult to make.

A similar condition of the lung, with similar physical signs, is produced by *hypostatic congestion or œdema*, such as we are familiar with in the course of morbus cordis; but, as a rule, this occurs only when the patients are very ill, have been long in bed, and lying more or less constantly upon the back. Besides this, the affection is bilateral. In the same way a solid mass of *new-growth* or a *hydatid* in the lung might simulate a localised pleuritic effusion, but the diagnosis of these affections is not always so difficult as might be anticipated, because the area of dulness is either in an unusual place or of an unusual shape.

In many of these cases where the physical signs are misleading, the history of the patient and the general condition will make the diagnosis certain. For example, with a pneumonia, however puzzling the physical signs may be, the high fever, the sudden onset, and course of the disease, and the altered pulse respiration ratio help to make the diagnosis clear.

In a second group of cases the difficulty arises because with an effusion, instead of the voice- and breath-sounds being greatly diminished or absent, they are exaggerated, so that sometimes even well-marked bronchial breathing is heard. In many of these cases, if all other physical signs fail, the character of the dulness may give the correct diagnosis; for it is extremely rare, under any other conditions, to get that peculiar, absolute tonelessness or stony dulness on percussion which is so common with an effusion. If, in a case of doubt, the ordinary signs of displacement of the organs be present, there can be no doubt as to the diagnosis, for such displacement hardly ever occurs except with effusions. On the left side, even with effusions of moderate size, the area of stomach resonance is diminished; this is a point of some importance, but it is not quite conclusive, for there are cases of fairly large effusions, in which the area of stomach resonance is not only present but sometimes even increased. This is more common in children, but it occurs also in the adult.

These difficulties are only likely to arise with serous effusions at the base of the lung; in other parts of the pleura a localised effusion would in all probability be purulent, and there would be other symptoms which would point to the diagnosis of localised empyema.

Certain conditions of the chest walls, *inflammatory tumours*, *new-growths*, and other *affections of the chest walls* are not always easy to distinguish from pleuritic effusion.

The most difficult cases of this group are those of *localised abscess in the chest walls.* These are usually what are called "cold abscesses," of a very chronic character, and of a tubercular nature. These cases will be further discussed when empyema is being dealt with, for it is with a pointing empyema that they are most likely to be confused.

Pericardial effusion rarely offers any real difficulty in diagnosis, for the area of dulness is continuous with that of the heart, and does not extend far enough to the back. Even when there is a *localised empyema close to the heart*—a very unusual place, be it observed—the peculiar irregularity in the shape of the dulness will be very suggestive of the correct diagnosis. Cases of this kind will be also referred to again under empyema.

Certain conditions below the diaphragm.—The conditions that offer the greatest difficulty in diagnosis are those in which there is some disease beneath the diaphragm pushing it up into the thorax; for instance, *subphrenic abscess*, *abscess hydatid or cancer of the liver*, *affections of the kidney and spleen*, or *an abscess which tracks upwards* from below.

On the left side the difficulties in diagnosis are not so great as they are upon the right, nor so frequent.

I have seen a case of abscess due to gastric ulcer on the left side, which caused so much displacement of the diaphragm upwards that the diagnosis of empyema was made, but exploration, repeated on more than one occasion, was fruitless. All the symptoms pointed to the thorax rather than to the abdomen, and the diagnosis was not established until the autopsy was made.

On the right side, owing to the presence of the liver, the shape of the area of percussion dulness is important; thus the lower border, whether the disease be in the liver itself, or between the liver and the diaphragm, will stand lower than it should, indicating that the right lobe of the liver is either pushed down or enlarged. The upper line of dulness rises with a bold curve into the thorax, in the nipple or mid-axillary line, and the line is not, as it is in cases of effusion, straight in front and rising to its highest at the back. The upper border is often raised in greater proportion than the lower border is depressed, and it is often roughly stated that increase of the dulness upwards means either hydatid abscess or cancer in the liver, or hydatid or abscess between the liver and diaphragm.

In most cases of general peritonitis, or ascites, if the diaphragm be displaced, it is thrust up into the thorax, carrying the organs with it more or less symmetrically, and no difficulty is presented: but this is not always so.

Thus I remember a case of ascites in which the right side of the chest was dull up to the third space in front, while on the left side the dulness was not above that which is usually met with in ascites. The right pleura was tapped high up in the fifth space in the mid-axilla, the diagnosis being made of a large pleuritic effusion, but it was the abdomen that was emptied through this puncture. After death the diaphragm was found to be displaced, so as to correspond with the line of dulness described, and the pleura was healthy. In this case fluctuation could be obtained in the upper intercostal spaces in the axilla by tapping the abdomen. This, I think, might have suggested the correct diagnosis, for I have certainly never seen fluctuation obtained so high up as this, transmitted through the diaphragm from fluid below it to fluid above it. If I had another case of this kind I should tap the abdomen first.

Pneumothorax, *with effusion*, rarely causes any difficulty in the diagnosis; yet it is often missed, because not thought of; but if it be remembered, the characteristic signs give the diagnosis at once.

One difficulty that constantly arises in pleuritic effusion is to determine the condition of the upper parts of the lung upon the side affected, this being of importance in the bearing it has upon the cause of the effusion, for any affection of the apex would suggest that the effusion was of tubercular origin. Unfortunately a large effusion alters the physical conditions of the apex to such an extent that the physical signs cannot be relied on. On the one hand, the lung, being compressed and congested, may yield some exaggeration of the breath sounds with some prolongation of expiration and crepitation, even when the lung is otherwise healthy. On the other hand, even when the lung is phthisical, the characteristic signs may not be obtained, owing to compression of the air-tubes and interference with the entry of air into those parts; and so it happens that with a large effusion tubercular disease of the apex might exist and not be recognised, or, on the other hand, be diagnosed when the lung was healthy. For these reasons it is best to wait until the effusion has disappeared before any decided opinion is expressed upon that point. Of course, if bacilli have been found in the sputum, the diagnosis is then made certain, but otherwise not.

The diagnosis of the size of an effusion.—This, of course, is a relative term, and depends upon the amount of fluid in relation to the size of the chest; thus a few ounces in a child may produce almost as marked symptoms as pints do in the adult; but in any case the actual size of an effusion is very difficult to guess at, beyond a general statement that the effusion is large or small; thus I have drawn 150 ozs. from the chest when I did not expect to get more than half that amount, and I have failed to get more than 40 ozs. from a large adult when the chest seemed to be brimful of fluid.

THE TREATMENT OF PLEURAL EFFUSION.—During the early stages the treatment of Pleuritis Serosa is the same as that of Pleuritis Sicca. The patient should be kept warm in bed, placed upon a febrifuge or mild diaphoretic mixture, be given a mild aperient when necessary, fed upon light food, and have the affected side gently counter-irritated.

Under simple treatment of this kind, the inflammation comes to an end, the fluid is rapidly absorbed, and convalescence established in two or three weeks' time.

Counter-irritation.—When the fluid is larger in amount, more powerful or continued counter-irritation may be employed. Thus mustard poultices might be frequently applied; or the side be frequently rubbed with some rubefacient liniment, like the Lin. Terebinth. Acet.; or a lint or spongiopiline jacket may be made and freely sprinkled on the inside with turpentine or spirits of camphor.

Iodine applied to the skin is a favourite remedy, partly because of its action as a counter-irritant, and partly because of its absorbent action, like that of iodide of potassium when given internally, upon the pathological products of inflammation.

There is a choice of three preparations of iodine, according to the action desired, viz., the tincture 1 in 40, the liquor 1 in 20, and the liniment 1 in 8.

With sensitive skins, as in women or children, and especially in little children, iodine, even in the weakest form, acts as an irritant, and cannot be long used, because of the pain it produces and the inflammation of the skin it excites. The liniment frequently blisters, even in an adult, and is on that account not so often used.

Blistering.—When a blister is required, it is best produced by brushing the side over lightly with tincture of cantharides or the blistering fluid of the pharmacopœia. Except in chronic cases, blistering is not desirable, but then it sometimes produces a rapid improvement when milder measures have failed.

Methods, especially directed to the removal of fluid by drugs, fall chiefly into two categories—the *derivative* and the *alterative*; but in estimating the effect of the action of drugs, it must be borne in mind that most cases of pleural effusion of moderate dimensions tend to get well of themselves if left alone without any treatment at all, or under almost any of the lines of treatment advocated.

Antipyretic treatment in any of its forms has been practically abandoned in the case of serous effusion, for it is not indicated, except where the temperature is high, and frequently the temperature is low, even with a large effusion.

Bleeding.—Venesection is quite out of place and is now abandoned, although it might find its use in certain cases where there is much cyanosis and dyspnœa, but would be then advocated because of the symptoms and not as a treatment of the disease. Nor are wet cups ever used now, although dry cups over a congested lung do certainly give relief. If a local bleeding be necessary, as it might be for pain, a few leeches will do all that is required.

Derivatives.—The treatment of serous effusion by derivatives is based upon the theory that the absorption of fluid from a serous cavity, like the pleura, can be produced by establishing a drain from the body from some other channel, for instance, from the skin, the bowels, or the kidneys. Thus, according to the predilection of the prescriber, diaphoretics, purgatives or diuretics would be employed. The fluids, however, thus discharged from the body are of a watery nature and not albuminous, and, so far as we understand the mechanism of the removal of fluid from the pleura, it is not by a process of absorption through the blood vessels, but more or less mechanically by a pump-like action through the lymphatics. The theory, therefore, upon which the derivative treatment of serous effusion is based seems to be incorrect; nor is there clinical evidence to prove that derivative treatment has the effect supposed. It has, moreover, disadvantages of its own, for, to be thoroughly carried out, it entails much discomfort and distress to the patient.

Diaphoresis.—Copious diaphoresis, whether produced by hot air or vapour baths, or by free administration of the more ordinary diaphoretics, is depressing. The most effectual diaphoretic, viz., pilocarpin, has several disadvantages of its own; for besides the depression which its full administration causes, it often produces a free discharge into the bronchial tubes, and may thus produce severe dyspnœa, or greatly aggravate dyspnœa if it already exist.

At the same time, a mild diaphoretic mixture is excellent during the febrile stage, and adds greatly to the comfort of the patient, but it is given on general grounds and not with a view to the removal of the fluid.

Purging.—Drastic purges produce great exhaustion, and, if pushed, upset the digestion and destroy the appetite, and so do more harm than good. At the same time it is advisable to keep the bowels gently and regularly moved, and for this purpose some mild purgative will be probably necessary.

Diuretics.—There is no conclusive proof that diuretics as such promote the absorption of fluid, and accordingly Scoparium, Squill, Juniper and such-like diuretics are out of place. The most useful drugs recommended as diuretics are Digitalis and Citrate of Caffeine; but they are really useful, not because of their action as diuretics, but for the effect they produce upon the heart.

Another line of treatment, based upon similar theories, is that of the so-called *Thirst-cure* or *Dry Diet cure.* The principle of this is the restriction of the fluid which the patient takes to starvation limit. This method of treatment, though warmly advocated, has been frequently tried, found wanting, and abandoned. It causes great discomfort and suffering to the patient, disturbs the digestion, and makes the taking of food a difficulty.

Alteratives.—The treatment of serous effusion by means of alteratives, such as mercury, iodine and salicylate of soda, has more to recommend it.

Mercury and Iodine.—Mercury and iodine, especially on account of the influence they have in promoting the absorption of the pathological products of inflammation, might be expected to prove useful; while all three have a decided bactericidal action when taken into the body, and might therefore prove useful in this way also, knowing, as we do, that so many of the serous

effusions are of bacterial origin. These remedies may be applied externally, in the form of the tincture liniment or liquor of Iodine painted on, or of an ointment of Iodine or of Mercury, or a combination of the two, such as the Iodide of Mercury.

Internally, *Iodide of potassium* is one of our stock remedies, frequently combined with quinine. A prolonged course of mercury, in cases of tubercular pleurisy, may do good in the same way, as it is stated sometimes to do in cases of acute tubercular disease of the lungs ; but for acute cases it is not much used. It is in place chiefly for chronic cases of considerable duration, and then its action is difficult to estimate.

Salicylate of soda was introduced chiefly on the theory that pleurisy is commonly of rheumatic origin.

This subject has been already discussed, and it has been shown that pleurisy is by no means a common complication of rheumatic fever, and that the evidence which attributes ordinary cases of pleurisy to rheumatic fever is probably quite incorrect.

I have used Salicylic Acid and Salicylate of Soda frequently in cases of pleurisy, but have not been able to satisfy myself that they were of any use.

The best line of treatment for cases of moderate effusion is the following :—

1. During the febrile stage to keep the patient warm and quiet in bed, and feed him upon such food as his digestion can assimilate.
2. To relieve any symptoms that may present themselves, for instance, pain and cough, by the appropriate means, which need not be further specified.
3. To give the patient a mild diaphoretic mixture ; to keep the urinary secretions active by the use of an ordinary febrifuge mixture ; and to keep the bowels sufficiently moved.
4. To maintain the general strength by food, and by such drugs as digitalis and caffeine when necessary, or by general tonics such as quinine.
5. To use iodide of potassium, or perhaps mercury, to promote absorption.
6. To apply warmth and counter-irritation to the affected side, best of such intensity only as to keep the vessels continually dilated, rather than to cause any profound alteration in the nutrition of the skin, by the use of such remedies as Spirit of Camphor, Turpentine, Capsicum, or the mild preparations of Iodine. A more powerful counter-irritant, such as the stronger preparations of iodine and blistering, are only in place in chronic cases, and after the febrile stage is passed.

After the febrile stage is passed and the temperature has been normal some days, the patient may be allowed to get up, but great care should be exercised. He should not be allowed to leave his room for some days later, nor to go out of doors at all, except in suitable weather, for a chill very often leads to a relapse.

If such common-sense treatment of the affection be carried out, assisted by such treatment as the nature of the case or the special symptoms may suggest, most cases get quite well without trouble, and without more than the necessary amount of discomfort or suffering to the patient.

It is important to remember this fact in connection with the next line of treatment which we are going to discuss, viz., that by paracentesis.

PARACENTESIS.

Paracentesis has become so much the routine treatment of serous effusion that no doubt it is very often performed where it is not really necessary ; and for any long series of cases that would show the natural history of the affection, *i.e.*, the prospects of cure without paracentesis, we have to go back to years ago, when the operation was not so common.

Thus, Louis gives a series of 229 consecutive cases of serous effusion, out of which 224 got perfectly well without paracentesis.

Only 1 of this number died, and that was the result of pericarditis. A similar series of cases has been published by Walsh.

Paracentesis is, when properly carried out, such a perfectly simple and safe operation that, with proper care, it cannot do any harm. Yet statistics, such as those quoted, are of interest and importance as showing that all the good results which follow paracentesis are not necessarily to be credited to the operation.

No arguments are now necessary in favour of paracentesis; all that is necessary is to consider the details of the operation, and when it is desirable to perform it.

1. *Urgent cases.*—There are certain cases in which paracentesis is not only the right thing, but the only thing to be done. This is when the effusion is large and the symptoms severe.

Then it may be necessary to tap the patient as soon as he is seen without a moment's delay. This may be called "*Paracentesis Necessitatis.*" Urgent symptoms do not depend upon the size of the effusion only, but to a great extent upon the rapidity of the development of the fluid.

Thus very large effusions may be discovered where there is little in the symptoms to indicate their size; and, on the other hand, some effusions not of very large size may be associated with severe symptoms, especially if the fluid has developed rapidly. The urgency, of course, will be greatest where effusions have formed, or re-formed after paracentesis, with great rapidity.

Thus paracentesis may become urgent within a day or two of commencement of illness, and may have to be repeated in a very short time.

For instance, a young man of about 25 years of age was admitted on what was the third day only of illness. His chest was brim full. Paracentesis was urgent; 80 ounces were removed, and in three days' time he had to be tapped again, and the same amount was removed, after which he made a rapid recovery.

I have also removed 127 ounces after only ten days' illness, so that the rate of effusion may be very rapid.

On the other hand, small effusions sometimes cause such grave symptoms as to require paracentesis before they reach any large size. This is not common with simple effusion, but is more likely to occur when the effusion comes as a complication of some other affection, *e.g.*, in the course of phthisis, pneumonia, or morbus cordis; or, again, where the effusion is double, *i.e.*, on both sides. In all these cases delay is dangerous, paracentesis should be performed at once, and if the fluid re-accumulate, repeated also without delay, even before the symptoms again become severe.

2. In another class of cases paracentesis, though not urgent, is desirable, and that without much delay, *e.g.*, where the effusion is very large, although there may be no severe symptoms produced by it at the time. In such cases it is unlikely that the fluid will spontaneously disappear, for, owing to the pressure (if to no other cause), the pleural pump is out of gear and cannot work until the pressure is relieved; and though in time the relief may come spontaneously, still it will be only after lapse of some time, and all that while the patient is liable to sudden aggravation of symptoms, which would bring the case into the preceding group and make paracentesis urgent.

With large effusions the removal of even a part of the fluid may lead to the rapid disappearance of the rest.

3. A third group of cases is formed by those in which the effusion is of moderate dimensions, and in which no important symptoms are produced by it.

In nearly all these cases, as stated, the effusion will in all probability spontaneously disappear in time. What we have to consider is whether paracentesis will accelerate cure, and when it should be performed. Upon these points there is room for great divergence of opinion. Some advocate the earliest possible interference, even as soon as the effusion can be diagnosed. Others would leave it for a period of two or three weeks, and some still longer.

As to early interference, it is very difficult to prove the advantage of interference by figures, which are as likely to mislead as to lead to a right conclusion. There seems to be a general consensus of opinion, with which my personal experience agrees, that it is not desirable to perform paracentesis too early, during the ingravescent stage, unless the effusion reach large dimensions rapidly or severe symptoms be present. As long as the effusion is of moderate dimensions it is best to postpone paracentesis till the active or acute stage of the disease is passed, or at any rate until the case has been watched for some little time.

Most of the cases at St. Bartholomew's Hospital which were tapped had been ill, though not in the hospital, for two to three weeks. In some cases paracentesis was performed at once, being urgent, and in many after only two or three days' stay in the hospital.

On the other hand, experience is equally strong in favour of not leaving the effusion too long unrelieved, and it is generally felt that if an effusion shows no sign of diminution by the end of the third week, it will be well to tap.

The reasons for early paracentesis are chiefly theoretical.

First, that the effusion is checked. Of this there is no conclusive evidence. Some authorities, indeed, maintain that operation during the active stage excites effusion, and acts as an irritant to the pleura. At any rate, what is constantly observed is that the fluid is rapidly reproduced after paracentesis, just as if no good had really been done.

Secondly, that the longer the lung is left compressed by the fluid the more likely it is to be bound down by adhesion, so as to become incapable of re-expansion. Now, although there are a few cases recorded in which extensive adhesions have formed within a few days, such cases are altogether rare. If adhesions do form so early, they are usually soft and unresistant, so that they offer no real difficulty to the re-expansion of the lung when the inflammation has subsided and the fluid has been removed or has spontaneously disappeared. In the great majority of cases, even when the fluid has been left for weeks, the lungs come out again without difficulty when the fluid is withdrawn, and there are many cases recorded in which the lungs have re-expanded completely after being compressed by an effusion for many months.

Thus, Woillez records a case of complete re-expansion of the lung after an effusion of nine months' duration, and I have seen the same occur after an effusion of double that duration.

Thus the two main indications for early paracentesis proving to be based on theory rather than on clinical experience, it follows that there is no need to be in a hurry to perform paracentesis, but that it may be safely postponed for two or three weeks, or even longer if it should be thought desirable. At the same time it should not be postponed too long; for there can be no doubt that when the acute ingravescent stage is passed, recovery is greatly accelerated; that is to say, much time is gained by the removal of the fluid even when the effusion is small.

We might therefore sum up the question of paracentesis in this way: There is no reason to hesitate to perform paracentesis whenever it seems in any way indicated; at the same time there is no necessity to be in a violent hurry if the symptoms do not suggest it.

The **general frequency** of paracentesis is about 50 per cent., *i.e.*, in cases of pleuritic effusion nowadays about 50 per cent. will be tapped. Of the 200 cases from St. Bartholomew's Hospital 92 were tapped, equal to 46 per cent. Of 50 cases under my own care, 27 cases were tapped. Of the latter, 2 were tapped twice, and 3 three times. This agrees exactly with the larger figures from St. Bartholomew's Hospital, in which 10 per cent. of the cases required more than one paracentesis.

Contra-indications for Paracentesis.—I do not think there are really any, but there are a few questions which deserve consideration in this relation.

(i) *Fever.*—In many cases that come under observation the temperature is normal, for the effusion has existed some little time and the febrile stage is passed. In acute cases, where the temperature is still high, the operation may be performed safely, if necessary, but the temperature is not usually materially affected in any way by the operation. It is quite unusual to see the temperature drop after paracentesis for serous effusion in the way it often does after paracentesis for empyema, yet it may.

Usually the fever continues for a time much as it was before paracentesis, even when the effusion does not re-form. (*Cf.* Chart III., p. 671.)

Of course the persistence of high temperature shows that the inflammation of the pleura has not completely subsided.

It may even happen that paracentesis may be followed by a rise of temperature in a case in which, at the time of operation, the temperature was normal. This is probably to be explained by the irritation of the pleura, caused by the two layers coming once more into contact, for in many a return of pain and friction occurs.

(ii) *Phthisis.*—So many more cases of pleurisy are of tubercular origin than was formerly supposed as to suggest the conclusion, which I think to be correct, that the tubercular origin of an effusion does not affect the question of paracentesis at all.

There is a theory, it is true, that pleural effusion checks the progress of phthisis in the lungs. I do not think that this rests upon any reliable clinical evidence, and I certainly do not agree with it. Within my own experience this theory has been responsible for leaving effusions unrelieved for a long time, and yet in the end paracentesis has been followed by complete recovery, without any progress in the disease in the lung.

Pleural effusions associated with phthisis may therefore be treated in the ordinary way, but as the lung is already damaged, care will be necessary if the aspirator be employed, for too great suction may easily cause the diseased lung to rupture.

(iii) *Purulent Transformation of the Fluid.*—More used to be heard of this risk in years past than is heard nowadays, for in the first place this transformation was not infrequently seen, and, in the second place, it was believed to be the natural course of a serous effusion which lasted any length of time. This theory is, however, wrong, and we now know that serous and purulent effusions, depending, as they do generally, upon different pathogenic organisms, are as a rule serous or purulent from the commencement, and remain so till the end. The purulent transformation of a serous effusion means fresh infection with pyogenic organisms. This infection might arise spontaneously from within, *i.e.*, from the lung or organs within the chest, or be introduced from without by paracentesis.

In the latter case it is due to dirty instruments or a careless operator, and if the ordinary antiseptic precautions be observed, the risk of converting by paracentesis a serous effusion into an empyema may be practically disregarded.

Many years ago Dieulafoy[1] maintained this in opposition to some of his contemporaries, and his results could hardly be excelled by those of recent years. In 180 punctures in 69 cases of pleuritic effusion there was no instance of purulent transformation. I remember to have seen such transformation occur not altogether infrequently in my student days, and occasionally also since, but I have never had an instance of it in my own practice, so that I believe such a result to be entirely within our own control.

[1] *Bull. de l'Académie de Méd.*, 1892, xxvii. 488.

I have seen also the opposite occur, viz., a purulent fluid become less and less purulent as paracentesis was repeated, and end at last by becoming serous, when the patient rapidly recovered.

Operation.—An anæsthetic is unnecessary, for the pain of puncture is very slight. It is also on its own account undesirable, for an anæsthetic deprives the operator, during the removal of the fluid, of the guidance which the patient's feelings otherwise give.

If, on account of fear or excitement, some anæsthetic be thought necessary, a whiff of nitrous oxide gas would be sufficient; at any rate ether should be avoided on account of the irritation it causes to the air passages.

A pad of lint, soaked in a 20 per cent. solution of cocain, and applied for an hour or two before the operation is performed, will remove the pain of the puncture through the skin, but it will not affect the pain felt as the needle passes through the intercostal spaces, especially if it strike the ribs. A small subcutaneous injection of cocain would make the operation absolutely painless; but injections of cocain are not devoid of risk, and may give rise to unexpected and unpleasant symptoms. Really the pain is so trifling that it is best for the patient to resolve to endure it without any anæsthetic.

The aspirator is in most cases quite unnecessary, and, if employed, should be only exhausted to such an extent as to make the fluid flow. The great objection to its use is that unless a manometer be attached (and this does not form part of the ordinary apparatus), it is impossible to say what amount of suction is being employed, especially towards the end of the operation. Too much suction may cause the lung to rupture, and this is the common explanation of pneumothorax occurring during paracentesis.

Rupture of the lung is less liable to occur with serous effusion than with empyema, for the lung is not so likely to be diseased near the surface; yet it is impossible, in a case of pleurisy, to be certain that the lung is sound; and if, by undue suction, a phthisical cavity be opened, its contents will be sucked into the pleura, and with them very possibly putrefactive organisms, which will convert the serous into a purulent effusion.

Thus, in a man of 50, 4 ounces only could be removed. The aspirator was then used; the lung burst, and in a few days' time a fœtid empyema formed, which required resection of the ribs. In the end the patient made a good recovery, but only after some weeks' illness.

Even if the lung be sound, there may be adhesions which bind it down in places, so that as the fluid is removed, irregular expansion will take place, and in this way even a healthy lung may be stretched to such an extent as to give way.

I doubt if any healthy human being could stand a pressure of more than 9 inches of mercury, and many will hardly stand more than 3 or 4. The pressure of a few inches of mercury is quite sufficient to burst a healthy lung out of the body, so that the dangers of the aspirator, when carelessly used, are by no means imaginary.

The safest apparatus for tapping serous effusion consists of an ordinary trocar and cannula, with a tube extending to the floor. The tube, when filled with fluid, acts as a syphon.

Usually there is pressure enough in the pleura to fill the tube with fluid; but if not, the syringe may be necessary before the syphon can act.

The end of the tube should be placed in a small vessel filled with water, so that no air may enter the pleural cavity, if it should so be that the intra-thoracic pressure is negative. I have seen air gain access to the pleura in this way; but, of course, if the mouth of the tube be under water such an accident cannot occur.

Under this arrangement the fluid flows away under a negative pressure of from 18 to 24 inches of water; that is, roughly speaking, the distance from the chest to the floor. This is equivalent to about $1\frac{1}{2}$ inch of mercury. The fluid, as long as it flows, will remain under the same constant negative pressure, and not as when the aspirator is used under a negative pressure which constantly varies, from almost zero to even several inches of mercury.

The effusion should not be removed too rapidly, and therefore the needle selected should not be a large one. Usually the middle-sized needle supplied with the aspirator, of about $\frac{1}{16}$ of an inch in diameter, is large enough.

The Place of Puncture.—This should, of course, be as far as possible in the centre of the dull area; but as in the majority of the cases of serous effusion the fluid occupies the lower part of the pleural cavity, the common spot for puncture is on the horizontal level of the nipple in the fifth or sixth intercostal space in the mid-axillary or posterior-axillary line. A position lower than this is not so convenient, for as the fluid flows the diaphragm quickly rises, and reaching the mouth of the cannula, may choke it.

Mode of Operation.—The needle should be held firmly in the hand at right angles to the chest wall and pressed, with firm and constant pressure, slowly inwards. Directly the prick of the needle is felt, the patient will shrink and change his position. It will then be difficult to know where the point of the needle is, and it will almost certainly strike the rib. If this happen it will cause the patient pain, and may turn or break the point of the needle. If the needle be still pushed on, it may slip over the edge of the rib and enter the chest with a jump, and thus go farther than was intended or desired.

To avoid this, before the needle is used the finger or thumb of the other hand should be firmly pressed down into the intercostal space at the place where the puncture is to be made, and the needle then passed over the centre of the nail. By this means, even if the patient shrink, the place will not be lost, and the needle will be easily introduced through the space and will not strike the ribs.

Easy as it may be thought to avoid the ribs in paracentesis, it is in practice by no means so easy as it seems.

The amount necessary to be removed.—There is no need to limit this by any theoretical consideration. If a syphon tube be used, I should recommend that as much be removed as will flow away easily. How much this will be in any given case it is impossible to foretell; for even with large effusions it may happen that little can be removed on account of unpleasant symptoms which arise; but if the pleura can be emptied, or nearly so, so much the better. I make it a rule to take away as much as I can remove in this way without discomfort to the patient, and I have never had reason to think this general rule wrong. It is true that the removal of a small amount of fluid is often followed by the spontaneous disappearance of the rest, but this cannot be calculated upon, and if the pleura has been only partly emptied and the fluid re-form, paracentesis will become necessary all the earlier; besides, it is a good thing to get the lung to expand as freely as it can without risk or discomfort.

I have occasionally, after emptying the pleura as far as I could with the syphon, affixed the aspirator to try how much more I could get away under a greater suction. Frequently I have failed to get more than an ounce or two, and that with the production of distress to the patient. Thus, after removing 30 ounces with the syphon, a suction of −30 inches was necessary to drain 10 ounces more, and this caused much cough and pain in the chest, and was all that the patient could bear.

It follows, therefore, that all the fluid that can be safely removed can usually be taken away with the syphon alone, and that the aspirator is not necessary.

The amounts removed, of course, vary. Sixty ounces is a fair average, but it may greatly exceed this.

Any amount over 100 ounces is uncommon.

Among the 250 cases analysed, 105 ounces were removed once, 112 once, 115 once, 120 three times, 127 once, 130 once, 139 once. Besides these cases I have twice removed 150 ounces.

The largest amount recorded is, I believe, in a case of Liebermeisters,[1] viz., 7 litres, equal to 245 ounces. When the paracentesis has to be repeated, the amounts removed usually decrease on each tapping, but not always.

	1st Puncture.	2nd Puncture.	3rd Puncture.	Rate of Formation.
1.	100 ounces.	40 ounces.	10 ounces.	...
2.	63 ,,	94 ,,	...	...
3.	50 ,,(6th day)	50 ,, (5 days later).	27 ounces.	10 ounces a day.
4.	24 ,,	60 ,,	43 ,,	...
5.	90 ,,	100 ,, (14 days later).	...	6 ,,
6.	50 ,,	57 ,, (4 days later).	...	14 ,,
7.	70 ,,	90 ,,	40 ounces.	...

Some of the recorded amounts appear almost to be incredible and impossible.

Thus Hodges[2] evacuated 13 pints (260 ounces) at one operation; and Oge,[3] after removing 4 pints on one day, removed 14 more within forty-eight hours, *i.e.*, 360 ounces in two paracenteses.

Of pus even larger amounts have been removed. *Cf.* Wilson Fox, note, p. 967, *e.g.*, 11, 15, and 20 pints.

Risks of the operation.—There are practically no risks at all if ordinary care be used and if the diagnosis be correct.

Of course it is in all cases important to ascertain carefully, before the operation is commenced, where the heart is placed, and this may be determined by auscultation, if by no other means.

The diaphragm is very unlikely to be touched if the needle be inserted in the place recommended and directed towards the middle of the chest. A mere puncture of the diaphragm is, however, of no serious consequence, and I have even known, in a case of ascites, the abdomen emptied through a needle inserted into the right chest, to remove an effusion which was thought to be there and was not, so that the diaphragm was penetrated.

Nor is the lung likely to be injured. It is, I suppose, often pricked, especially if the needle pass into the chest with a jump. A mere puncture will do no harm, and a laceration of the lung by the needle, I believe, hardly ever occurs.

If the lung be torn during paracentesis, it is generally not due to the needle but because the aspirator has been used and too much suction employed.

Wounding of the intercostal artery is a danger more imaginary than real. I have never seen such an accident occur, though a few cases of it are recorded.

Indications for the stopping of paracentesis before the complete removal of the fluid.—The indications are mainly these—pain, cough, general distress, and faintness, change in the character of the fluid withdrawn, and dyspnœa. These symptoms are most likely to arise when the fluid has been removed rapidly, and especially if the aspirator be used with too much suction; still they may occur in any case, and with every precaution.

[1] *Deutsch med. Woch.*, 1890, No. 11.
[2] Boston, *Med. and Surg. Journ.*, 1869.
[3] *Practitioner*, vol. xii.

1. *Pain.*—This is usually felt after the fluid has been flowing for a short time; commonly beneath the upper part of the sternum, under the clavicle, or in the shoulder, and occasionally underneath the lower part of the sternum or in the præcordial region.

It is evidently often severe, for the patient may become very restless with it or even feel faint, and the pulse may become unsteady.

Under such circumstances the flow of fluid should be stopped for a time, and a little brandy given. When the patient has recovered the flow of fluid may be started again. Sometimes a good deal more fluid may be removed before pain is again experienced, but usually the pain increases directly the flow of fluid re-commences, and soon becomes so severe that the operation has to be stopped.

No doubt, in most cases, the pain is due to the stretching of adhesions, which may even be felt by the patient to snap. As soon as the operation is over, the pain usually ceases. It may last sometimes for an hour or two, and may then be relieved by a little stimulant and a few minims of opium, or by strapping the side firmly.

2. *Cough* is sometimes very troublesome, and is perhaps the commonest cause of the stopping of the operation. It is dry, short, frequent, painful, and sometimes almost paroxysmal. It usually commences after a certain amount of the fluid has been withdrawn, and becomes worse if the withdrawal of fluid is continued.

It may be due to the irritation of the lung, as it expands, by the cannula, and will then diminish if the needle be slightly drawn out; this manœuvre, at any rate, should be tried before the operation is stopped. But it is often to be attributed rather to irritation in the lung, consequent on its re-expansion and to the alteration of the circulation through it.

If the cough be very severe, and if it continue after the needle is removed, a little tincture of opium should be given on the tongue and the side be firmly strapped for a few hours.

3. *General Distress.*—Sometimes, without either pain or cough, the patient complains of great general discomfort and alarm, and passes into an emotional, almost hysterical, condition, and soon begs that the operation may be stopped, saying that he cannot endure it any longer. Yet the attacks are not hysterical, for in nervous, emotional patients who dread the operation the excitement is present before the operation is commenced, and generally ceases as soon as the needle has passed and the fluid flows; whereas, in these cases the condition comes on after the fluid has been flowing for some time, and often in patients who have had no dread of the operation, and are phlegmatic rather than nervous, excitable persons.

The attacks are very like what is seen in certain cardiac conditions, and they may very likely be of cardiac origin, consequent on the disturbance of the circulation by the removal of the fluid from the pleura. At any rate, they have a real basis, and may prove of considerable gravity if disregarded.

It is best at once to stop the flow of fluid, and if the symptoms do not pass off quickly to terminate the operation.

4. *Change in the character of the fluid.*—

(*a*) *The fluid may become blood-stained.*—This is due usually to the rupture of some vascular adhesion, or to the tearing of vascular vegetations on the surface of the lung. It is usually developed towards the end of the paracentesis. It is rarely more in amount than enough to stain the fluid red, the colour being bright red, like that of recent blood, not dark, as in those cases where the fluid itself has been sometime hæmorrhagic. In those rare cases in which the intercostal artery has been wounded with the needle, the hæmorrhage may, of course, be free.

In small amounts it is of no significance, and need not interfere with the completion of the paracentesis; but if it be more than a little it is well to suspend the operation.

(*b*) *Air may pass with the fluid.*—This means that the lung has been ruptured, and should be an indication for stopping the operation at once; for although, if the lung be healthy, the mere entry of air into the pleura will do no harm, still if a cavity have been opened it may lead to the passage of the contents of this cavity into the pleura, and the conversion of a simple serous effusion into an empyema.

5. *Dyspnœa.*—This sometimes sets in unexpectedly, and may quickly become considerable; but it is quite a rare complication.

If the patient complain that the breath feels short, the operation should be suspended for the time; the sensation then, as a rule, quickly passes off, and the operation may be completed. It is probably more subjective than real, and depends upon the alteration of the circulation through the lungs.

In some rare cases dyspnœa may be considerable and attended with some degree of cyanosis. The cause of this is often obscure, but it is sometimes connected with another rare and remarkable occurrence during paracentesis, viz., albuminous expectoration.

6. *Albuminous or serous expectoration.*—It sometimes happens that during the paracentesis the patient begins to cough and complain of some shortness of breath, and soon after to expectorate a quantity of clear frothy fluid. The cough is almost constant, though not very violent or paroxysmal, and with it there is some shortness of breath and occasionally even considerable dyspnœa.

Wheezing and crepitation are heard over the lung, usually over the affected side only, but occasionally on both. After the symptoms have lasted for an hour or two, or perhaps a little longer, they usually subside, and the case runs its ordinary course, but every now and then the symptoms are extremely severe, and the patient dies of suffocation.

Albuminous or Serous Expectoration.

Albuminous expectoration is really very rare. Terillon[1] collected 21 cases, chiefly of French origin. Since that time isolated cases have been recorded, but the total number is still quite small, probably under 50. The figures often quoted give a quite erroneous idea of the real frequency of the affection.

Thus Ewald[2] states the disease to have occurred in his practice in 1 out of 26; Martineau in 1 out of 50; but its frequency is really very much less than this. I have seen but one instance of it out of a very much larger number of cases, and I believe it to be quite one of the rarest events in pleuritic effusion.

My own case occurred in a man of about 40 years of age, who had a right-sided effusion for about three or four weeks. I performed paracentesis myself, using the syphon and not the aspirator. The fluid flowed readily, and after about 40 ounces had been withdrawn the patient began to cough; the cough increased in frequency and caused him much distress. This was soon followed by a little dyspnœa. Expectoration commenced in about ten minutes, and in an hour the patient brought up 8 ounces. The attack lasted for three hours, during which a pint of frothy fluid in all was coughed up. Over the right side, that is the side of effusion, there was a good deal of wheezing and crepitation, and a little also on the left. The patient's condition was in nowise such as to cause alarm; the symptoms rapidly subsided, and the patient made a good recovery.

The cases differ a good deal *inter se.*

Access.—The attacks rarely come on during the paracentesis, but subsequent to it and after an interval, which may be a few minutes only, and is rarely more than an hour; though in a few instances it has been as much as two hours.

[1] *Thèse de Paris,* 1873.

[2] Wilson Fox, references, p. 1070.

The duration of the attack is usually short, not more than an hour or two, but occasionally longer; in the longest recorded case the attack continued for two days and two nights (forty-eight hours).

The *quantity of fluid* varies usually with the duration, from a few ounces only to even more than 3 pints. The actual amount often appears much greater than it is, owing to the number of air-bubbles with which it is mixed.

In character the fluid is frothy, in appearance like that of acute bronchitis. On standing it divides into three layers, the upper whitish and very frothy, the middle opalescent, the lower denser and more viscid. Analysis shows that it is rich in mucin and poor in albumen, the opposite being the case with the fluid of the effusion.

Analysis of Fluid (Terillon).

The pleural effusion.		The albuminous expectoration.	
Ppt. with acetic acid . . .	Slight.	. . . .	Copious.
Mucus (*Terillon*) . . .	1·6 per cent.	. . . .	1·4 per cent.
Albumen (*Dujardin Beaumetz*)	6 to 8 per cent.	. . . .	0·1 ,,

The *physical signs*, indicating congestion of the lungs, viz., wheezing and crepitation, are present usually on the affected side only, but occasionally on both; while in some of the fatal cases, though aggravated by the paracentesis, they were present before the operation was commenced.

There appears to be no necessary relation between the duration of the pleurisy prior to paracentesis and the liability to serous expectoration; but, as a rule, the cases have been of at least some weeks' standing.

Where paracentesis has been repeated, the serous expectoration has sometimes followed each operation, and Behier [1] records a case in which this happened four times.

It is *not to be connected simply with the use of the aspirator* or the employment of too much suction in drawing the fluid off, for in 16 (non-fatal) cases, recorded by Dieulafoy, the aspirator was used only in 4, the fluid in the other 12 being removed by syphonage; while in 6 fatal cases the aspirator was used only in 3. Dieulafoy,[2] however, thinks that the risk is increased if the fluid be drawn off in too large quantity and with too great rapidity.

The *result* is rarely fatal, though the symptoms may be very alarming for a time.

Complications and post-mortem appearances.—Among the fatal cases some other complication besides the effusion is generally found, *e.g.*, morbus cordis, pleural adhesions over the opposite lung, a clot in the pulmonary vessels, or, as in Scriba's [3] case, a fibrinous plug in the bronchus of the affected side. The general *post-mortem* appearances of the lungs are those of extreme œdema.

Serous expectoration is stated, moreover, *not to be peculiar to paracentesis.* Thus, cases are described, in the course of empyema, of thoracic aneurysm; and in some cases of pleural effusion it occurred before paracentesis. Similar expectoration may occur when the trachea has been pressed upon and on the point of being perforated by an aneurysm or new-growth; but as the fluid is then saliva-like, not mixed with air, and frothy, these cases of tracheal pressure can hardly, I think, be fairly compared with the conditions we are now discussing.

[1] *L'Union Médic.*, 1873.
[2] *Bull. de l'Acad. de Méd.*, 1892, xxvii. 488.
[3] *D. A. f. klin. Med.*, 1885, xxxvi. 328, and for other references.

Serous expectoration certainly seems to be *less frequent of recent years* than it was twenty or thirty years ago, and I think this must be connected with the earlier performance of paracentesis.

Theories.—There are three explanations of the phenomenon which have been suggested.

I. *Perforation of the lung,* and the discharge of the fluid from the pleural cavity through the lung; the perforation being due either to laceration by the needle or to rupture by the aspirator.

To this theory there are several insuperable objections.

1. That, if the result of perforation, the discharge of fluid ought to commence at once, and not after an interval, sometimes a long interval, as is occasionally the case.

2. That air should escape with the fluid during the operation, and the physical signs of pneumothorax develop, but this does not occur.

3. That spontaneous perforation of the lung, common as it is in empyema, is practically unknown with serous effusion.

4. That a puncture made by the needle would be too fine for the fluid to escape by. Puncture of the lung is certainly not a rare accident in paracentesis, and produces no results of consequence. The argument that punctures are not found *post-mortem* in the fatal cases is not worth much, because the aperture is not always to be found even in pneumothorax.

5. That when the effusion is large, as it usually is in the cases where albuminous expectoration occurs, the lung is so far away from the seat of puncture that it could hardly be reached by the needle.

6. Lastly, there is the difference in the chemical constitution of the two fluids, viz., that in the pleura and that discharged by expectoration, the former containing much albumen, and the latter much less albumen, and a great deal of mucus.

II. *Absorption of the effusion by the lung.*—The difference in chemical constitution of the two fluids is also conclusive against this theory, to which may be added the further objection that the passage of the fluid into the lymphatics of the lung through the stomata is the natural process by which fluid is removed from the pleura, and that, however rapid this may be, it is not accompanied with the exudation of fluid from the bronchial tubes.

III. There remains the third and only satisfactory theory, viz., that the condition is the result of *œdema of the lung.* This theory seems to conform best to the facts, for—

(*a*) The condition takes some little time, it may be an hour or so, to develop.

(*b*) In the fatal cases œdema of the lung is the pathological condition found on *post-mortem* examination, and with this the physical signs observed during life agree.

(*c*) In some cases œdema of the lung has been diagnosed during life and found *post-mortem*, although there had been no albuminous expectoration.[1] The simple explanation, then, is that death occurred so rapidly that sufficient time had not elapsed for expectoration to occur.

(*d*) Theoretical considerations support this view; for when a lung has been for some time collapsed and is rapidly distended, the result must be that not only will air pass into the air vesicles, but blood into the blood vessels, as well as lymph into the lymphatics, just as happens in the subcutaneous tissue after the application of dry cups to the skin. Cohnheim showed that the permeability of the vessels in a lung which had been some time collapsed was far greater than that of a healthy lung, so that the fluid would pass more readily from the blood vessels into the lymphatics, and probably also from the lymphatics into the air vesicles or bronchi.

But though these general conditions, favourable to the production of œdema of the lung, exist in every case which is tapped, yet serous expectoration is a very rare event, and some explanation must be found for the fact that it does not occur under ordinary circumstances. Some nervous influence, leading to paralysis of the vasomotor nerves, has been suggested as an explanation. "Obscurum per obscurius" is the best criticism with which to meet such a theory as this, for it rests on mere speculation and is unsupported by facts.

There is more ground for the belief that the explanation is to be sought in abnormal pathological conditions. In the non-fatal cases the serous expectoration must depend upon some transient condition which soon rights itself. It seems most natural to connect this with the sudden disturbance of the circulation to which the withdrawal of fluid must lead, a view which is supported by Cohnheim's observation just referred to. This would be most marked and most

[1] Woillez, *L'Union Méd.*, 1873, No. 77. [2] Herard, *ibid.*, No. 86.

likely, therefore, to produce symptoms when a large amount of fluid had been withdrawn rapidly ; and it may be due to the improved methods of paracentesis that the condition is less frequent now than it used to be.

In the fatal cases the phenomenon, though excited by the withdrawal of fluid, must depend upon some abnormal condition which is persistent. Thus, if the blood gain access to the lung freely through the arteries and yet cannot circulate, owing to some obstruction either on the side of the veins or lymphatics, œdema of the lung would necessarily occur, and conceivably also a discharge of fluid, just as swelling of the leg occurs when the lymphatics or main veins of the limb are obstructed. The morbid changes found *post-mortem* support this view ; thus in some instances morbus cordis has been found, in others a clot in the pulmonary vessels. Lastly, there is the remarkable case recorded by Scriba, in which the main bronchus was found plugged by a fibrinous coagulum. The condition of the lymphatics has not, so far as I know, been carefully investigated in these cases.

Sudden death in connection with paracentesis.—This is the rarest of all occurrences in pleuritic effusion.

If one can judge from recorded cases, it is probably rarer now than it used to be, because in the present day effusions are not allowed to remain so long, or to reach so large a size without relief.

It may occur at three different periods—either immediately on the introduction of the needle, during the operation, or shortly after the operation is over.

In the first case it is due to shock, and is similar to what is seen in puncture of the abdomen. Instances of this very rare event are recorded by Besnier and also by Leichtenstern.

Sudden death occurring during the operation, or soon after it, is to be referred, no doubt, to the same causes as lead to that event before the effusion has been tapped, or to the complications which have just been described as the result of the paracentesis, viz., œdema of the lung and albuminous expectoration.

In the 250 cases upon which my statistics are based, in 2 only did death follow shortly after paracentesis, but in neither did the fatal result depend, as far as could be seen, upon the operation. In the first, a man of 48, the operation was performed successfully, though only 20 ounces of fluid were removed, and then only with the help of an aspirator. The next day the patient suffered much from dyspnœa ; in the evening, thirty hours after the paracentesis, he expectorated a bronchial cast 6 inches long, and died of suffocation. No autopsy was permitted, but as some patches of membrane were observed on the fauces, it is possible that the patient had diphtheria.

In the second case, a man of 35, a large effusion existed, which was relieved by paracentesis. Subsequently, after getting up, the patient had a relapse, fluid re-formed rapidly, and was again removed by paracentesis. Thirty-six hours later severe dyspnœa set in, and the patient died in a few hours. The necropsy (beyond 36 ounces of fluid in the pleura) showed nothing but a flabby and somewhat fatty heart. The condition of the heart was, no doubt, the cause of death.

Physical signs after paracentesis.—Usually as the fluid is removed the lung expands and the organs return to their normal places. Breath- and voice-sounds become audible in parts where they were absent before, and frequently friction is heard and may be felt, showing that the lung has reached the chest walls, and that the two layers of the pleura are actually in contact.

Friction is most frequently heard in the upper part of the chest, or, at any rate not in the lowest. It may be audible over nearly the whole side. Usually it disappears in time, as the two layers of the pleura become adherent, but it may continue a long time. I have heard it nine months after the paracentesis, as plainly and as extensively as at first.

With the return of breath sounds wheezing or crepitation are also heard, and are no doubt due, in most cases, to some exudation of fluid into the air-tubes ; the crepitation may be the result of the opening out of the collapsed air vesicles, and is then not easy to distinguish from some forms of pleuritic friction. In either case these physical signs quickly pass off.

The percussion over the upper parts of the chest often becomes markedly tympanitic, so as to suggest the occurrence of pneumothorax; but as the voice- and breath-sounds are audible, and often, in addition, crepitation and wheezing are heard, while the more characteristic signs of pneumothorax are absent, it is clear that the tympanitic resonance is connected with the expansion of the lung. This is to be explained, I believe, in the same way as that met with above a pleural effusion or a pneumonic lung, as due to the loss of elastic tone or tension in the lung, the nutrition of which has been impaired by being for some time collapsed.

This hyper-resonance after paracentesis is generally met with in the upper part of the chest in front, but it may occur elsewhere; for instance, I have seen it behind, at the angle of the scapula. It usually passes away in a few hours, but it may persist for two or three days. With this hyper-resonance is sometimes associated great exaggeration of the breath-sounds, so that they may become bronchial or even amphoric. This, too, is a transitory phenomenon.

Pneumothorax after paracentesis has been already referred to. It is usually due to rupture of the lung owing to excessive suction being employed with the aspirator. Sometimes, when the intra-thoracic pressure is negative, air may enter by the needle, for which reason it is wise, if the syphon be employed, that the mouth of the tube should be kept under water; but more commonly, if air enter the thorax through the needle, it is later in the operation and after a certain amount of fluid has been removed.

It has been stated that air may gain access through the skin puncture at the side of the needle. I do not think this is possible, and I certainly have never seen it happen.

The presence of air in the pleura after paracentesis has also been referred to the exhalation of gas from the fluid. That the effusion contains much gas, which can be easily sucked out of it, is seen in the aspirator-bottle, while it is being exhausted. This, however, only occurs when the bottle already contains air, and does not happen if the mouth of the tube by which the exhaustion is being made is below the level of the fluid, or if the bottle be completely full of fluid.

In the same way in the pleura the exhalation of gas from the fluid implies the pre-existence of air already in the pleura, but it does not explain how the air got there.

The presence of air in the pleura is not in itself harmful, as long as it is aseptic, for it is readily absorbed. The risk is, that the air in entering the pleura may carry infective organisms with it, and excite some fresh or different kind of inflammation. The source of infection is no doubt to be found in the tubes used, if they have not been properly cleaned and disinfected, and it is during its passage back through them that the air acquires its infective properties.

In certain cases disinfected air has been deliberately introduced into the pleura, with the object of displacing the fluid by air, which may be more readily absorbed.

The cases in which this procedure has been recommended are those in which the lung is bound down by adhesions so that it cannot completely expand to fill the pleura, the space left becoming in consequence refilled with fluid, almost as soon as it has been emptied. When used in this way the air should be passed through boiled water containing carbolic acid, or some other antiseptic, and filtered through cotton wool.

Experience does not, as a matter of fact, show any material advantage for this method of treatment over that of leaving the fluid there, and performing paracentesis when necessary.

Indeed, the air is quickly absorbed, and the fluid returns just as if no air had been introduced. At the same time, this is a method which might be reasonably tried in obstinate cases.

Results of paracentesis.—The results of paracentesis, when properly carried out, are almost always beneficial. In some cases recovery is very rapid, and a single paracentesis may be sufficient for cure. Even the removal of quite a small portion of the fluid is often followed by a rapid absorption of the rest. Frequently the operation has to be repeated (10 per cent.). No special rules can be laid down as to when, and how often, paracentesis should be performed; but, speaking generally, the indications for its repetition are the same as those for the original operation, except that it is not necessary to wait so long. There are, in my experience, no objections to repeated paracentesis, and though some writers set their authority against it, I cannot understand their reasons now that the operation is so free from risk when properly performed.

Treatment by Free Incision.

If paracentesis fail to cure an effusion, is there anything else that can be done? The only thing that remains is to lay the side freely open and to put in a drainage tube; in fact, to treat it in the same way as an empyema. Whether this is a line of treatment which can be safely adopted, will depend upon the nature of the particular case in question. It will, of course, be quite unsuitable to any case where pleuritic effusion is secondary to some grave disease, as, for instance, advanced phthisis or mediastinal tumour; and it is practically restricted to cases which are, as far as can be estimated, of simple origin, or, if of tubercular nature, where there are no signs of progressive disease.

The objection is that, do what we will, even with the strictest antiseptic precautions, it will be found practically impossible to prevent the serous effusion from being converted into an empyema; and although the risks of empyema may not be so great as we have been hitherto accustomed to regard them, still they are certainly greater than those of simple serous effusion.

There are but very few cases recorded in which this method of treatment has been adopted. I have treated two cases in this way, and both with success.

1. A young lady, aged 31, whose abdomen and right pleura had been full of fluid for more than twelve months, came under my care with the question as to whether anything could be done to relieve her. The fluid had been known to be present for some months, but paracentesis had never been performed, either of the pleura or the abdomen. The case was one evidently of tubercular origin, but there were no signs at the time of any progressive mischief; for although the patient was extremely thin and feeble, yet there was no elevation of temperature or night sweats, no cough or expectoration, nor anything to indicate the presence of phthisis.

The abdomen was tapped, and the fluid did not return. The pleura was tapped repeatedly, in all thirty-nine times in the course of twelve months. At the end of that time the side was laid freely open with the usual antiseptic precautions, and a drainage tube inserted. The day after the operation the temperature rose; it became hectic, and continued hectic for a considerable time. A week or so after the operation, the fluid, which had hitherto been serous, became purulent, but the discharge was never copious, and the drainage was free, so that the fever did not seem to be due to the condition of the pleura, and it was feared that a fresh outbreak of tuberculosis had occurred. For three months the patient remained in a very critical condition, but then began gradually to improve; the fluid ceased to be purulent, soon the discharge ceased, and the side was closed, the lung expanding completely, and no external deformity was left. It is now more than seven years ago; the chest still continues, I believe, perfectly normal, and no fresh signs of any kind have developed, either in the lung or in the pleura, the recovery

being, so far as the pleura goes, complete. Fluid returned after some time in the abdomen, and this has required paracentesis on several occasions, but in other respects the patient is well.

Shortly after the publication of this case, Dr. Morrison recorded a similar one which had been under his care in the year 1882.

Of this case the following is a short summary:—

A woman, aged 23, suffering from ovarian tumour, was found to have a right pleural effusion. The abdomen was tapped, but it filled again. The pleura was then tapped, in all six times, at intervals of a few days, the quantities removed being 25 ounces on the first paracentesis, 90 on the second, and 20 on each subsequent occasion. Then the side was opened, and a drainage tube inserted. For five days after the operation the discharge was profuse, the temperature remaining at 100, as it was before the operation. On the sixth day the discharge suddenly ceased; on the eighth day the tube was removed; and in fourteen days the side appeared to be perfectly well. A few days later ovariotomy was performed, but the patient never rallied from the operation. No *post-mortem* examination was made.

Somewhat later a paper was read before the Clinical Society by Dr. Wilson, upon three cases in which the pleura was incised for serous effusion within a few days of the onset of the disease. In the first two cases, the patients were well after about ten days. In the third case, which was complicated with influenza and pneumonia, the patient was almost moribund at the time of operation. For some weeks subsequently the temperature continued to rise at night, and some pus was discharged. About three months later, the wound, which had been allowed to close, was reopened, and about 4 ounces of pus escaped. Drainage was then effectually provided for, and in the end recovery became complete in about nine months after the operation.

2. My second case is the following:—Alfred Y. came under observation on 21st September 1896 with right pleural effusion of thirteen months' duration. The side was brim-full, measuring $1\frac{1}{2}$ inches more than the other ($17\frac{1}{2}$ to 16), and the heart was greatly displaced. Exploratory puncture showed the fluid to be serous, and cultivation proved it sterile.

Paracentesis was performed on 26th September, and 38 ounces removed; again on 23rd October and on 17th November, with the removal of 80 and 47 ounces respectively. The fluid reaccumulated, and it was decided to make a free incision. This was done on 6th January 1897, in the axillary line in the seventh space, and a drainage tube inserted; a large amount of fluid was evacuated. A piece of rib was not excised; it was not thought necessary at the time, and did not become necessary afterwards.

The day following, the temperature, which had been previously normal, rose to 102°, P. 120, R. 40.

For a few days much fluid escaped, but it remained serous, gradually decreasing in quantity for five days, when it became purulent, at the same time diminishing very greatly in amount, so that not more than 2 ounces were discharged in the twenty-four hours.

On 13th January, *i.e.*, a week after the operation, the following note was taken:—

"The patient seems comfortable and well, and is suffering no pain. The respirations are quiet and number about 20. The wound is healthy."

The side was now explored with a probe. It was found that the lung had expanded in all directions, and nothing seemed left of the pleural cavity except the long track in which the tube lay. In most parts this was quite narrow, not more than half an inch, and probably less, in diameter; but at the end, about 8 inches from the opening in the side, it was somewhat wider, and measured about an inch in diameter.

The percussion note in front in the upper part, down to about the level of the upper border of the third rib, was hyper-resonant, the breathing somewhat amphoric in character, and a bell-sound obtained. Possibly some air was present in the pleura here, but if so, the cavity must have been cut off from the lower part. In a few days this hyper-resonance disappeared, and with it the bell-sound.

The patient soon became well, except for the fistulous track in which the tube lay.

On 9th March the following note was made:—

"The probe shows that there is a long tubular sinus, half an inch in diameter and 6 inches in length, extending down in the direction of the spine, straight inwards from the incision in the chest wall. The vocal vibrations are felt all over the right base behind as distinctly as on the other side, but there is not much respiratory murmur heard here. In front the breathing can be heard feebly over the whole upper part of the chest down to the level of the nipple."

On 18th May I examined the side somewhat carefully, to see if a further incision could be made in order to drain the small bulbous cavity in which the fistulous track seemed to end. This, however, was found to be so small, and to lie so close to the bodies of the vertebræ, that it could not be safely reached with the knife. The patient was then sent away to the country.

In August the patient was *in statu quo*, and was taken into St. Bartholomew's Hospital with the object of seeing if anything further could be done. The sinus was practically unchanged. The pus amounted to about 3 to 4 ounces in the twenty-four hours, but seemed more than the sinus alone could account for. Yet repeated exploration with a probe failed to discover any pouch or cavity.

The pus was examined bacteriologically, and found to contain a small number of tubercle bacilli. The sinus was injected daily with creasote, dissolved in sterilised oil (25 per cent. solution). About 3 ounces passed in, and were left in for two hours. Beyond a little smarting, no discomfort was caused, and the patient seemed to improve. After the first injection or two the patient complained of slight headache, and the urine became a little smoky, but these symptoms passed off without altering the treatment, and did not recur.

The injections were continued for a month and then abandoned, for though the patient improved, no change took place in the sinus or the amount of discharge.

From this time to the present the sinus still remains much the same, but the discharge has become much less, not more than 1 ounce in the twenty-four hours.

The patient is well, and has for the last few weeks gone back to light work, which he has been able to do. He is still wearing a tube about a quarter of an inch in diameter and 2½ inches long.

The man died of phthisis subsequently, rather more than four years after the operation.

It should be noted in both these cases (1) that the effusion had been allowed to remain untapped so long in subservience to an old-fashioned and erroneous theory that effusion checks the development of tubercle in the lung; (2) that the lung, though so long compressed, was not bound down by adhesions, but expanded immediately after the fluid was withdrawn; (3) that what prevented complete cure was the fact that the sinus became itself tubercular or communicated with a tubercular cavity deep down in the chest; (4) that a portion of rib was not excised, and excision did not become necessary.

These cases show that the risks of laying the pleura open for serous effusion are by no means so great as we might *a priori* expect, a conclusion which is confirmed by the experience of the result of surgical injuries to the thorax. Although the free incision of the pleura is a perfectly justifiable operation in appropriate cases, it does not, however, follow that a large serous effusion should be treated by incision in preference to paracentesis. The results of paracentesis are so satisfactory, and recovery after it is often so rapid, that the question of incision need hardly ever be raised; and the operation ought to be reserved only for those cases in which paracentesis has been tried repeatedly and failed. That incision of the side may be safely performed in such cases is an important addition to our knowledge, and I cannot doubt that it will be more frequently practised in the near future; but that is a very different thing from suggesting incision as the routine treatment of serous pleurisy as it is for empyema. We know, of course, that a careful surgeon, with strict antiseptic precautions, may safely, and often with advantage, open an inflamed knee-joint. At the same time, surgeons, so far as I know, do not recommend that opening of the knee-joint should be the routine treatment for simple synovitis. The pleura is certainly as important a serous cavity as the knee-joint, and should be treated with at least the same amount of caution.

The After-treatment of Pleural Effusions.

The general after-treatment is directed to restoring the patient to good health by means of change of air, good food, and tonics. It may be wise to send the patient to a warmer climate for a winter or two, if that be possible, especially where there is a suspicion of tubercular disease. Many cases do quite well in this country, if care be taken of them; but, under any circumstances, care will be necessary during the cold and changeable months of the year. At the same time, there is not much danger of relapse, especially if the pleurisy has been a severe one, for generally the pleuritic cavity is completely obliterated. I have, however, known of one or two instances in which a second effusion has taken place upon the previously affected side.

Special treatment will be directed towards getting the lungs to expand completely and removing any contraction of the side which may have resulted from the pleurisy. For this purpose gentle gymnastics are useful, especially such calisthenic exercises as will expand the chest without unnecessarily violent effort.

In the same way, walking, rowing, and to some extent mountain climbing, are useful; but the exercise must not be overdone, and must in all cases stop short of actual fatigue.

These aids may be supplemented by methods more especially directed to the lungs. The patient, for example, may be caused to expire under increased pressure, and for this purpose various forms of apparatus have been devised, such as Waldenberg's gasometer, and others. A simple method, which I have used with advantage, is to cause the patient to breathe through a fairly large tube, the mouth of which is placed at the bottom of a tall jar or jug filled with water, so that expiration is made under a pressure of about 18 to 24 inches of water.

In dealing with the defects left behind by pleuritic effusion, the essential factor in all methods of treatment is time. Time alone will lead to cure or great improvement, without any special methods of treatment at all, for, at the best, these are but auxiliary. With stiff joints it is surprising what time and patience, combined with gentle use, will do, and so it is also with the pleura. It is better to trust to time and nature than to have recourse to violent and forcible measures, which, though they may sometimes seem to accelerate recovery, in a large number of cases delay it and do more harm than good.

63. PURULENT EFFUSION—EMPYEMA.

It has already been stated that there is no absolutely certain means of diagnosing the nature of a fluid effusion short of obtaining a specimen of it by the needle. Till that has been done the diagnosis is but an opinion, with more or less of probability in its favour. In what is to follow the diagnosis of empyema is assumed to have been established.

The onset of empyema may be sudden, with shivering, pain in the side, and rapid breathing, very like what occurs in pneumonia, and it is probable that many of these cases with acute onset are really of pneumonic origin.

In other cases the access is insidious. There may be a little pain in the side, a little cough and some shortness of breath, but nothing very definite to direct attention to the chest.

Occasionally empyema is found when there have been no special symptoms at all, or where they have been masked by those of some other disease, *e.g.*, phthisis, scarlet fever, typhoid fever or pyæmia. Both these latter groups have been described as *insidious* or *latent empyema*.

Fever.—The fever is usually of a hectic character, like that which accompanies suppuration in other parts of the body, the temperature rising to 102° or 103° every evening, and being at its lowest in the morning. The inverted type, viz., that in which the morning temperature is the highest, is occasionally met with as in other cases of hectic fever.

The daily oscillations are considerable, the difference between the maximum and minimum often being as much as several degrees. These oscillations may be seen even when the maximum temperature is quite low, and when during a great part of the day the temperature is subnormal. This may be compared with what occurs in the post-febrile stage of some acute fevers, notably typhoid, influenza, and pneumonia.

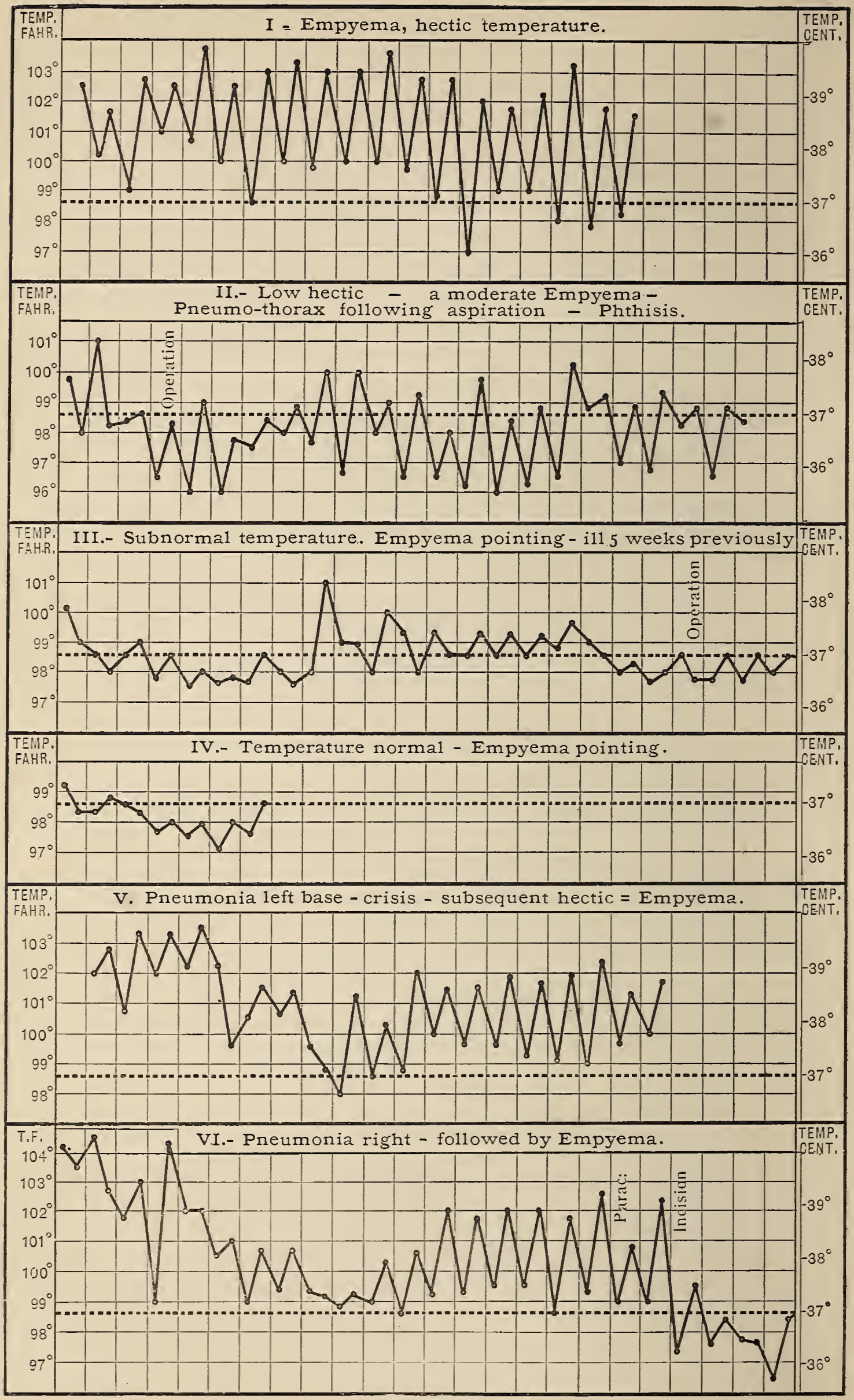

I = Empyema, hectic temperature.
TEMP. FAHR.
TEMP. CENT.
103°
102°
101°
100°
99°
98°
97°
39°
38°
37°
36°
II.- Low hectic — a moderate Empyema — Pneumo-thorax following aspiration — Phthisis.
Operation
101°
100°
99°
98°
97°
96°
38°
37°
36°
III.- Subnormal temperature. Empyema pointing - ill 5 weeks previously
Operation
101°
100°
99°
98°
97°
38°
37°
36°
IV.- Temperature normal - Empyema pointing.
99°
98°
97°
37°
36°
V. Pneumonia left base - crisis - subsequent hectic = Empyema.
103°
102°
101°
100°
99°
98°
39°
38°
37°
VI.- Pneumonia right - followed by Empyema.
T.F.
104°
103°
102°
101°
100°
99°
98°
97°
Parac.
Incision
39°
38°
37°
36°

Fig. 150.

The temperature is sometimes found to be normal, and that when the empyema is still developing and active, so that fever is not a necessary accompaniment of suppuration in the pleura, any more than it is of suppuration in the peritoneum, pericardium, brain, or indeed in any other part of the body. This fact is of great importance in respect of diagnosis, for it shows that the absence of fever is not conclusive against the purulent nature of a pleuritic effusion.

Whether the temperature has been normal or subnormal from the commencement, *i.e.*, whether the empyema has been afebrile throughout, is a difficult question to answer; for many cases of empyema, especially in hospital practice, do not come under observation until they have been ill some days, or, it may be, weeks. I cannot remember to have observed any case of empyema from the beginning in which the temperature was not raised during the first ten days at least, even when it rapidly fell to the normal subsequently, so that many of these afebrile cases may be really only cases in which the febrile stage was short.

Of the 156 cases analysed, the temperature was normal as long as the case was under observation in 7 instances; in some of them the empyema was actually pointing; one of them was a case of empyema associated with cancer; and there were two instances of what I have called low hectic, the charts of which are given.

Where empyema follows pneumonia, the temperature, which may have fallen to the normal or nearly so, rises again and becomes hectic as the empyema develops. As already stated, this recurrence of fever may give the first indications of this complication. A difference in temperature on the two sides of the body is sometimes observed, that on the affected side being a degree or two higher than on the sound side. It is a rare phenomenon, and, though of interest, has no clinical importance.

The Constitutional Signs are usually well marked. The patients become pallid, feeble, and ill, lose flesh, sweat much during sleep, and have occasional attacks of chilliness, sometimes of actual shivering; but distinct rigors are not common.

The constitutional symptoms stand in close relation with the fever, though not invariably, and both are usually well marked in the early stages of the disease and subside or disappear subsequently; but the constitutional signs may be marked and suggest suppuration even where the temperature is normal. In these cases it may be that a rise of temperature does occur at some time of the day, though it lasts only for a very short time, and can only be detected by keeping a registering thermometer strapped to the arm for several hours continuously. In one case of this kind the temperature was proved to rise daily to 103°, though only for about an hour.

The Physical Signs require no special consideration, for they do not differ essentially from those met with in other forms of effusion. There is perhaps only one which is conclusive, viz., *pointing*. When, over a spot in the chest walls, œdema develops, with redness of the skin and tenderness to the touch, we may conclude that pus is present and has made its way through the chest walls; soon a soft fluctuating swelling shows that the empyema is on the point of bursting. Pointing is never met with, except with purulent effusion, nor, I think, is local œdema. Slight general œdema, however, of the whole side occasionally occurs with any large effusion, whether serous or purulent, as well as with pneumothorax, and is to be explained by the compression of the intrathoracic veins due to the high tension in the pleura.

Short of local œdema, or actual pointing, marked bulging of the intercostal spaces is strongly suggestive of pus, for suppurative inflammation tends to involve

the adjacent tissues. In this way the nutrition of the intercostal muscles is affected, and their weakness allows the spaces to bulge. In serous effusion, although the intercostal spaces are widened, they do not bulge, except when the effusion is large and of long standing, in which case the nutrition of the muscles suffers by distension and pressure, just as that of the abdominal muscles does in chronic ascites.

The effusion varies much in *character* in different cases. It is generally homogeneous, of a creamy consistency and colour, and presents the usual characters of pure or laudable pus.

Sometimes it is thin and turbid, and on standing separates into two parts, a sediment of pus, and a supernatant turbid fluid. It is, in fact, sero-purulent, *i.e.*, a serous fluid mixed with a certain amount of pus.

This separation is said to take place in the thorax sometimes, but it can only occur when the fluid remains for some time undisturbed, that is to say, where the patient lies for some time quite still, and does not move the affected side even in breathing. It must, therefore, be rare. In these cases flocculent shreds are often floating in the fluid, which may choke the needle and cause difficulties during paracentesis. In other cases, only, I believe, in those of long standing, the effusion may be thick, even as thick as curds or clotted cream, so that its removal by the needle may be difficult or impossible. This condition is the result of inspissation or condensation of the effusion by the absorption of most of the fluid part; it represents an attempt at cure.

The colour is usually creamy yellow, but often has a greenish tinge. The presence of blood may make the colour pink or even red, but this is rarer with empyema than with serous effusion. Commonly the tint is salmon or terra cotta, or a dull, dirty, chocolate-brown.

The reaction is almost invariably alkaline, unless decomposition has taken place.

The smell is usually faint and sickly, sometimes sour or acid, and occasionally extremely fœtid. In the latter case the fluid has the dull or dirty brown colour, which shows the admixture of altered blood. Many of these differences are probably associated with different pathogenic organisms, and have therefore an etiological as well as a clinical value. They will be further referred to by-and-bye.

The size.—It is commonly stated that the largest pleuritic effusions are purulent, and there are no doubt instances on record in which really enormous amounts have been removed from the pleura by paracentesis. At the same time, in the present day the largest effusions commonly met with in practice are serous, probably because empyemata are recognised early and operated on.

The seat.—Empyemata may occupy the whole pleura, and amount to many pints, yet, as a rule, they discharge themselves either internally or externally before they reach such large dimensions.

Of small empyemata the usual seat, as with other effusions, is at the base, and that even when, as in apex pneumonia, the exciting cause of the empyema exists in the upper part of the chest, the pus gravitating to the lowest part and there exciting fresh inflammation.

Empyemata are more often localised than other effusions, and, when localised, are more likely to be found in peculiar places.

Conversely, when the signs of a localised effusion are present in a peculiar place, the probability is greatly in favour of empyema. Perhaps many of these localised empyemata have originated between the lobes of the lung, *i.e.*, have been interlobar, and thus have made their way to the surface in unusual places.

Where a localised empyema develops in close relation with the pericardium, the difficulties of diagnosis may be considerable. In one instance the dulness was continuous with the cardiac dulness, and extended a long way outside the nipple on the right side. The needle proved the presence of pus, and a free incision only made it certain that the effusion was in the pleura and not in the pericardium.

With these localised empyemata it may happen that the area of dulness may be extensive, yet the actual amount of pus be not more than an ounce or two, and contained in a cavity so small as to require great skill, and probably more than one attempt to reach it successfully with the needle. The extensive dulness is due either to consolidated lung round the pus, to a much thickened pleura or abscess-wall, or to some solid organ in close proximity.

The most difficult of the localised empyemata to diagnose are those in which the pus lies between the lobes of the lung or between the base of the lung and the diaphragm; for in these cases the physical signs often give but little assistance, and the pus may be very hard to find, even with the needle.

COURSE OF THE DISEASE.—The course of an empyema is usually steadily progressive, the amount of fluid increasing slowly but continuously.

Purulent effusion rarely develops with the rapidity often seen in serous effusion; this fact may be of some use in diagnosis, and very rapid increase in the size of an effusion will be in favour of serum rather than of pus.

Spontaneous Absorption.—If an empyema be left to itself, it is possible theoretically that the production of pus may stop and the effusion be absorbed.

The cells will then undergo fatty degeneration, and their products, mixed with the serum of the effusion, form a milky fluid, which will be absorbed. Sometimes among other disintegration products cholesterin-crystals are found in great abundance. Whether complete absorption can occur spontaneously or not is a difficult matter to prove satisfactorily; yet it certainly must occur after paracentesis not unfrequently, for there are many cases in which paracentesis has been followed by complete cure; and inasmuch as it is practically impossible to empty the pleura completely by paracentesis, it follows that the residue of the empyema must have been, and has in fact been, absorbed.

Partial absorption of a purulent effusion is a very common event. As a result of the absorption of the more fluid part of the effusion, the rest becomes inspissated or thick, and may have the consistency of curd or soft cheese. Such incomplete absorption occurs, probably as a matter of course, in every chronic empyema, for the pus in such cases is thick and curdy, and evidence of diminution in the size of the effusion is often given during life by the contraction which takes place in the affected side. In the course of time the absorption may be carried further, so that what is left is a firm, crumbly mass in which lime salts may be deposited. Specimens of this are to be found in all museums. As a rule, it is only when the amount of pus is small that complete absorption can occur. Yet some cases will be referred to later in which considerable amounts completely disappeared.

These results, though possible, are not common, and cannot be calculated on. As a rule an empyema, if left to itself, sooner or later makes its way out of the pleura. Generally it establishes a communication with the lung, and the pus is got rid of by expectoration, or it pierces the chest wall, and is discharged externally.

These results, which are the most frequent, are also the most favourable, for it is in these ways that many cases of empyema spontaneously cure.

But there is always danger in leaving an empyema to make its own way out, for the pus may take some other less favourable course, and by involving some other organ lead to grave complications.

Thus, in the thorax it may open into the pericardium, mediastinum, or some of the large vessels; or, piercing the diaphragm, may open into the peritoneum, or some adjacent organ, *e.g.*, the liver or colon; or, again, it may burrow and track in most irregular fashion, generally in a downward direction, and there is hardly any part of the abdomen or any organ contained within it which has not been known to become involved in this way.

Compared with perforation through the lung, or through the chest walls, any of these other results is comparatively rare.

Perforation through the Lung.—When a perforation through the lung has existed for some time it is easily found *post-mortem* as a small round hole, about an eighth of an inch in diameter, with thickened margins. The hole may be of larger size, but rarely exceeds a quarter or half an inch. It often leads by a somewhat sinuous track through collapsed lung tissue into a small- or middle-sized bronchus, but it rarely communicates directly with one of the larger bronchi. It may be situated in any part of the lung when the empyema is localised, but if the empyema be general, and the lung collapsed and flattened in the usual way along the spine, the hole is found somewhere in the middle third of the lung, often at the top of the lower lobe. There is usually one hole only, but occasionally there are two or three small ones, and sometimes many.

Thus, Woillez describes a case in which there were twenty holes in the lung; in the museum of St. Bartholomew's there is a specimen in which the pleura was riddled with holes twenty or thirty in number; but then the lung beneath was gangrenous.

It sometimes happens that in recent cases the hole is not easy to find, at any rate until the lung has been inflated under water.

Empyema does not, as a rule, burst through the lung until it has existed for some time, on the average eight to ten weeks, but it may not do so for several months. On the other hand, cases are recorded in which an empyema has discharged itself through the lung within two or three weeks of onset (13th day, Attrimont; 19th day, Trousseau).

Nowadays spontaneous perforation through the lung is comparatively rare, except where the empyema is localised or deep-seated, so as to be somewhat inaccessible; or where it has not been recognised and treated.

The imminence of rupture through the lung is often indicated by paroxysmal cough, which nothing seems able to control. The patient then coughs and coughs until completely exhausted. In such a case careful paracentesis is immediately indicated, and will relieve the paroxysm as nothing else will.

When perforation occurs, cough is at once excited and pus expectorated. The discharge takes place usually in mouthfuls, *i.e.*, in small quantities, a teaspoonful or so at a time; although, as the cough is almost constant, the amount brought up in a few hours may reach several ounces.

Profuse expectoration of pus, *i.e.*, the sudden discharge of a large quantity, is fortunately rare, for it is attended with great risk to life, and may cause sudden death from suffocation, just as profuse hæmoptysis may.

After the first few days the amount of discharge becomes less and less, and in favourable cases continues to diminish, until at last it ceases altogether. It is, however, quite unusual for an empyema to cure itself spontaneously in this way in less than several weeks, or it may be many months.

Occasionally it happens, especially in the more chronic cases, that the discharge occurs once or twice only in the day, and is then copious; the patient having a paroxysm of coughing, which lasts perhaps until half a pint of pus is expectorated, and then remaining fairly free from cough and discomfort, until the next paroxysm commences, which may not be for several hours. Such paroxysmal discharge of pus occurs especially in the morning after a good night's rest. It is excited no doubt greatly by change in position, just as it is in cases of chronic cavity in the lung, from which an empyema of this kind may sometimes be by no means easy to diagnose.

The quantity expectorated daily may be considerable, and continue for some time.

Thus Trousseau records a case in which 35 ounces of pus were expectorated daily for many weeks. In a case of my own nearly a pint of pus was expectorated daily, and at the same time an almost equal amount discharged by an external fistula; this had been going on for six months before the patient came under my observation.

It might be supposed that the passage of pus through the air-tubes for so long a period would cause grave complications in the lung itself. Strange to say it does not, and even after months the lung may remain perfectly healthy, and ultimately re-expand and return to its normal functions. This is remarkable, for it occurs with every form of empyema, even with those of a putrid character, and we must conclude, I suppose, that a kind of local immunity is produced, by which the lung ceases to be subject to the infection of, what would be to healthy lungs, virulent bacteria. At any rate, although inflammatory or septic complications in the lung may occur, they are certainly rare.

Nor even in tubercular cases is tubercle, as a rule, excited in the lung; and though in fatal cases tubercular changes in the lung are not infrequently found, still there is, in many of these cases, evidence to show that the lung changes preceded, and did not follow, the empyema, and were the cause rather than the result of it.

Although the connection between the pleura and the bronchus is so free that the pus is readily evacuated, still air hardly ever enters the pleura, and pneumothorax is but a very rare result.

This fact must be explained by physical conditions. It is thought to present considerable difficulties, and led Trousseau to conclude that pus could be squeezed through the vesicular structure of the lung as through a sponge, without any actual perforation at all; this view is, however, quite untenable. The real explanation seems to me to be this; *first*, that little or no movement takes place in the affected side or the affected part of the chest, and if it does, that the lung retains sufficient expansibility to respond to these movements; *secondly*, that the perforation is a fistulous tract and not a simple direct communication, as in the case of pneumothorax, and that it often passes in a more or less sinuous way through consolidated and condensed lung; for which reason also the discharge of pus occurs in small quantities at a time, and not in sudden gushes; *thirdly*, that this sinuous track is filled with pus which acts like a water valve of an inch or more in height, so that this amount of pressure at any rate would have to be overcome before the air inspired could force the pus back, and make its way into the pleura. Of course pneumothorax may occur, but it is rare, and then depends on exceptional conditions; such as the opening into the lung being of considerable size, or communicating directly with the trachea or one of the main bronchi. Perhaps for the same reason infection of the pus in the pleura by germs introduced from the lung is quite unusual, for an empyema discharging through the lung rarely becomes septic or putrid if not so to commence with.

The *prognosis* of empyema bursting through the lung is, as already stated, fair. Many of the cases—most, I think it may be said—get well; the statistics, however, are difficult to obtain. In former days, when free incision of the side was not often practised, spontaneous evacuation through the lung was regarded as greatly improving the prospects of recovery, and of better prognosis than a discharge externally through the chest walls.

Perforation through the Chest Wall.—When an empyema is going to burst through the chest wall, the intercostal spaces become inflamed, and the nutrition of the muscles suffers, so that the spaces bulge. When the pus has made its way through the fascia and muscles, and reached the subcutaneous tissue, the parts around become inflamed and œdematous. Soon in the centre a boggy spot is felt, in which fluctuation may be obtained; the skin over it becomes red, and, if not incised, gives way of itself.

The pus, when it has once reached the subcutaneous tissue, may burrow a long way and burst through the skin at points far distant from the hole in the intercostal spaces, so that the skin opening and that through the intercostal space do not correspond; indeed, there is usually a more or less long, fistulous and sinuous track, through which it is by no means easy to pass a probe into the pleura. Even on the thorax the external opening may be some inches from that through the intercostal spaces; and in some cases it may track still further on to the abdomen or back, and in one instance discharged itself through an aperture in the scrotum.

The common *seat of perforation* through the intercostal space is in the fifth or sixth intercostal space in the mid-axilla, but occasionally it may be more anterior, and Marshall stated that the most frequent seat was in the fifth intercostal space, just below the nipple, a spot less protected by muscle.

Trousseau stated that perforation was common between the ribs and the sternum, and for a similar reason, viz., the absence of muscle there. It is difficult, however, to see how the muscles can have anything to do with the perforation through the intercostal space, though they may influence that through the skin.

The skin openings may be numerous, for when the pus has infiltrated the subcutaneous tissues widely, the skin may give way in various places. Usually, however, one is sufficient to relieve tension, and as soon as the discharge is free the surrounding inflammation subsides and the abscess gradually closes.

Or if there be two or more openings through the skin, an incision will generally allow one of these to heal and result in a single track, through which the discharge takes place.

The opening through the intercostal space is much less frequently multiple, though it may be. Thus Lebert describes a case in which there were thirteen openings through the intercostal spaces. Two openings are not at all uncommon, and are often found in parts of the chest some distance from one another.

Thus I have seen a case in which one perforation was in the mid-axillary line low down, and the other in the second intercostal space, not far from the sternum, and a probe could be easily passed from the one to the other.

When the empyema is localised, perforation may occur in peculiar positions.

I have seen a case in which the opening lay immediately in the centre of the normal area of præcordial dulness. Where the heart may have been when the empyema burst I cannot say, for I did not see the case so early; but when the opening was established the heart lay immediately beneath it, though the pericardium itself was not involved, the discharge taking place round and over it.

A fistula, once established, may persist for a very long time without any other affection of the skin or of the rib. Caries of the rib is much less common after spontaneous rupture of an empyema than it is after incision and the wearing of a tube, no doubt owing to the pressure which the tube exercises upon the rib itself.

When an empyema discharges itself externally, weeks or months, as a rule, are necessary before recovery takes place. There are, it is true, instances in which cure has been effected in a short space of time, *e.g.*, a few weeks only. On the other hand, there are instances in which the fistula has persisted for many years.

I have myself had a case under observation in which the fistula had lasted for eighteen years; the man might possibly have lived much longer if he had been left alone, but an attempt at radical cure caused his death.

With an external opening, of course, pneumothorax is the rule, but where the opening is small it still may not occur.

An empyema may discharge both externally and also through the lung, as in the case I have already mentioned, and then enlargement of the external opening will usually allow that in the lung to close.

Perforation elsewhere.—Practically this is so rare as to be little else than a pathological curiosity.

(*a*) *Into other parts within the thorax.*—It has been stated that an empyema may communicate with some of the large vessels, both arteries and veins, but I can find no instance of this recorded. In the same way, it is stated that an empyema may burst into the pericardium. The pericardium may become involved as a complication of empyema, or a suppurative pericarditis may burst into the pleura, but I do not know of any instance in which the contrary occurred. A case is also recorded in which the vertebræ were eroded and pus found in the spinal canal, with all its consequences; but in this case it is probable that the spinal disease preceded the empyema, and was not caused by it.

The œsophagus may be perforated by it.

An interesting case of this is recorded by Voelcker,[1] in which two openings into the œsophagus existed, pus was vomited, and food passed into the pleura.

It might be thought that rupture into the mediastinum would not be so uncommon, and yet instances of it seem to be almost as rare as in the other cases, and when abscess of the mediastinum is found, it appears to be rather a complication due to the spreading of the inflammation than to actual penetration of the mediastinum from the pleura.

(*b*) *Through or round the diaphragm.*—It is not uncommon for abscesses below the diaphragm to perforate into the pleura, but the converse is rare. If this occurred it might cause sudden death by shock when the peritoneum was opened, or it might excite acute suppurative peritonitis, which would also rapidly prove fatal. Of the first I know no example, but of the second several instances are recorded.

Usually a local peritoneal, subphrenic abscess is produced, which may involve the liver or spleen, or open into the intestines.

In the latter case the pus would be passed by the bowels, and the communication probably be with the colon. It is possible that a communication might also be formed in the same way with the stomach and the pus evacuated by vomiting.

[1] *Clin. Soc. Trans.*, vol. xxiv. p. 86.

If the pus make its way posteriorly through the diaphragm, it may remain behind the peritoneum and burrow for some distance.

In this way it may simulate a psoas abscess, and point like a psoas abscess in the groin or even in the popliteal space. If the pus track towards the loins, it may present there as a fluctuating swelling, which has been known also to pulsate, and thus simulate an aneurysm.

THE COMPLICATIONS.—The complications of empyema fall into four groups, according as they are the result—

1. Of perforation.
2. Of the spreading of inflammation to neighbouring parts.
3. Of septic infection.
4. Of prolonged suppuration.

The bronchitis and congestion of the opposite lung, which occur in the course of a large effusion, cannot be considered as complications, for they are the more or less mechanical and natural pathological results of large effusion, and that whether the effusion be serous or purulent.

1. The complications which are the **result of perforation** have already been sufficiently considered. They are largely under control and preventable by proper treatment.

2. Those complications also, which result from the **spreading of inflammation** to the parts around, have already been dealt with under the general pathology of pleuritic effusion.

The only peculiarity of empyema is that the secondary inflammations may be, but are by no means necessarily, of suppurative character.

In the mediastinum suppuration is extremely rare, as already stated; but the most important result of the spreading of inflammation to this part is the fixing of the organs in the positions which they occupied when the effusion was at its greatest. This condition, by preventing the return of the heart and parts in the mediastinum when the effusion is removed, renders more difficult the process of cure, and greatly retards it; but even in extreme cases the adhesions will yield to some extent in the course of time.

The lungs.—Perforation of the lung and pneumothorax have been already discussed.

Acute inflammatory affections of the lung, as the result of empyema, are rare; for although pneumonia often enough leads to empyema, it rarely follows unless the empyema be of a septic character, and then the pneumonia is septic or gangrenous, and the result fatal. In some cases septic empyema excites suppuration in the lymphatics of the lung, chiefly beneath the pleura, which spreads thence some distance along the trabeculæ into the lung, following the course of the lymphatics, and leads to the condition which has been described as pneumonia-dissecans.

The secondary septic infections of the lung are invariably fatal.

The complication of most importance is that which depends upon the inflammation involving the pulmonary pleura, and leading to fibrous thickening of it, which may then form, as it were, a capsule over the lung, a quarter of an inch or more in thickness, and thus entirely prevent its re-expansion.

The Pericardium.—Pericarditis is fairly frequent, and occurs in about 13 per cent. of all cases. It is an anxious complication under any circumstances, and if the resulting inflammation be suppurative, invariably fatal. Fortunately the pericarditis is rarely of this character, but more frequently a simple fibrinous inflammation without even much serous effusion.

It generally quickly resolves, though adhesions are sometimes left, which may give trouble in the course of time. If suppuration occur and the condition be diagnosed, paracentesis or incision may save life, but in most of these cases the development of the effusion is insidious and the condition not recognised.

The *diaphragm* may become acutely inflamed, and in some cases its lymphatics are found stuffed with cells from the pleural to the peritoneal surface. Such an acute inflammatory infiltration must necessarily affect the nutrition of the muscle, and doubtless accounts in great part for the dyspnœa, which may be marked, even where the empyema is not of large dimensions. In later stages the diaphragmatic pleura is greatly thickened, and the connective tissue may extend to the interstitial tissue of the diaphragm itself, and even through it to the peritoneum on the other side.

This leads to a sort of cirrhosis of the diaphragm, with atrophy of the muscle cells, and to adhesions between its peritoneal surface and the organs in the neighbourhood, *e.g.*, the liver, stomach, or spleen. This change would, of course, be attended by more or less permanent loss of respiratory power, and would explain in part the dyspnœa, which is sometimes associated with chronic pleurisy, and which may be in excess of what the physical signs seem sufficient to account for.

The vessels and heart.—Although thrombosis occurs in empyema, as it does also in serous effusion, there is no evidence that this is due to the direct spreading of inflammation through the coats of the vessels; and in the case of the heart, except where pericarditis has preceded, it is to a septic infection that the lesions are to be attributed. The dislodgment of such a thrombus is the explanation of some of the cases of sudden death in empyema, whether after or without operation.

Empyema on the opposite side.—Empyema is sometimes double from the commencement, and many of these cases owe their origin to pneumonia. At other times a distinct interval elapses before the second side is attacked. In some instances we may presume that the inflammation has spread across the mediastinum from the one side to the other, but in the majority of instances the second empyema is the result of septic infection, of which, however, it may be almost the sole evidence.

3. **Septic Infection.**—This may occur in the three forms of *sapræmia*, *septicæmia*, and *pyæmia*.

The sapræmic form depends upon the absorption of toxic substances from the pleura. To this category may be referred the pains in the limbs and painful swellings of joints which are met with in the course of empyema, especially when fœtid. These pains are intermittent in character, and very obstinate to treatment; but they are often relieved at once by draining the pleura well.

The septicæmic form, generally due to streptococcus- or staphylococcus-infection, shows itself usually in the form of malignant endocarditis, associated often with myocarditis and pericarditis.

The pyæmic form is attended as usual with abscesses in various parts of the body.

Septicæmia and pyæmia are very rare as consequences of empyema, though empyema is common enough as the result of those affections.

There are also certain local conditions, suppurative in character, and no doubt infective in origin, which, though not uncommon, can hardly be placed in any of the preceding categories. I refer to secondary *empyema of the opposite side*, already mentioned, *abscess of the brain* and *meningitis*, to which may be added, as a much rarer complication, *parotid abscess*.

Intra-cranial complications.—The intra-cranial complications of empyema are cerebral abscess, cerebral softening, meningitis, thrombosis and embolism, all of them rare, and rarely occurring except in empyema of some long standing.

The association between these lesions and empyema is remarkable, because, though presumably septic and due to the carrying to the brain of infective matter derived from the lung or pleura (Boettcher has shown lung pigment in the contents of one such abscess), they are rarely associated with embolisms in other parts of the body.

Cerebral Abscess.—Among the causes of cerebral abscess, empyema takes a rank of its own, for in Gull and Sutton's [1] series 4 per cent. of all the cases of cerebral abscess were secondary to it.

The cerebral abscess is often single, but there may be several. The favourite seat seems to be the posterior parietal, temporo-sphenoidal, or occipital region.[2]

The abscess is often associated with meningitis, which is then usually suppurative, and may extend some distance over the base, or even down the spinal canal. It may also lead to thrombosis in the lateral sinus.

Extensive *cerebral softening* is sometimes the only lesion found in the brain, and this is, no doubt, in most cases the result of plugging of the vessels. It frequently affects the area supplied by the terminal branches of the sylvian artery, namely, the angular, supra-marginal, and the temporal sphenoidal convolutions, but, strange to say, in several recorded cases the occipital lobe on one side, or sometimes on both, was affected also, though the posterior cerebral vessels are but rarely the seat of thrombosis or embolism.

The softening is sometimes bilateral and symmetrical.

A female, aged 18, had a left-sided empyema,[3] from which two pints of pus were removed by paracentesis, and subsequently the side was opened and drained. A month later the patient lost the sight in the right eye and became blind for a time, but ultimately recovered the sight. A fortnight later she complained of defective vision in the other eye, and developed a weakness on the right side, which subsequently ended in complete hemiplegia. When she died, both temporo-sphenoidal, angular, and supra-marginal convolutions, as well as the occipital lobe, were in the condition of white softening.

The symptoms, as a rule, develop slowly, the earliest being severe pain in the head, which, after an interval, may be followed by fits, loss of power in the extremities, in the eye or face muscles, or by loss of vision.

In some cases the sudden onset of hemiplegia seems to point conclusively to *embolism.* Clots in the pulmonary vessels have been frequently described in long-standing empyemata, and are, no doubt, the source from which the embolisms, whether septic or not, are derived.

The following cases may be quoted in illustration :—

Case 1.—A woman, aged 46, had an empyema [4] which dated from parturition, four months previously. During the last three weeks before admission the chest was tapped three times, and 40 ounces, 12 ounces, and 80 ounces respectively removed. Three days before admission she became suddenly hemiplegic, with paralysis of the fifth and sixth nerves on the right side. She died two days later. On the *post-mortem* examination an abscess was found in the left temporal sphenoidal lobe, with pus in the left lateral ventricle, secondary suppurative meningitis, thrombosis with disintegrating clots in the whole left lateral sinus. There was a large amount of pus in the right pleura.

Case 2.—In another case of long-standing empyema with an external fistula, in which the pus was fœtid, two small abscesses were found in the brain, one in the middle of the left lenticular nucleus, and another a little farther forward. There had been no symptoms produced by these abscesses during life.

[1] Reynolds, *Syst. of Med.*
[2] Pye-Smith, *Path. Soc. Trans.*, xxviii. 4. *Cf.* Hadden, *St. Thos. Hosp. Rep.*, xvii.
[3] Handford, *Clin. Soc. Trans.*, 1888.
[4] West, *Lancet*, ii., 1885, 571.

Case 3.—In a patient who had had a discharging empyema [1] for three years, the cavity was opened and washed out. Two days afterwards the patient complained of severe headache; this was followed by fits, and a discharge of pus from the left nostril. After death an abscess was found in the left occipital lobe. The angular and supramarginal convolutions, as well as the occipital lobe, were in a condition of white softening, but there was no abscess or meningitis.

Incomplete hemiplegia following empyema of a transitory character, and ending in more or less complete recovery, has been regarded by some writers as of reflex origin. I cannot understand upon what grounds; and, from the fact that in most fatal cases, where the brain has been carefully examined, definite lesions have been discovered, I think there can be little doubt that there is an organic lesion in all the cases.

Brain abscesses occur after suppurative diseases of the lung, as well as of the pleura, *e.g.*, gangrene, chronic cavities and bronchiectasis, and they are often multiple.[2]

Speaking generally, septic infection of any kind which can fairly be regarded as secondary to the results of empyema is very rare, and becoming continually rarer, as empyema is recognised earlier and treated better, so that, to quote statistics of fifteen or even ten years ago would be misleading.

4. **Prolonged Suppuration.**—The result to which prolonged suppuration may lead is *amyloid disease*, but there is much in the causation of amyloid disease which is not yet understood. Thus in empyema it may come on early in the course of a few weeks in cases in which there is no large amount of suppuration, while in other cases, where the discharge is large, or even profuse, it may not develop for years or possibly not at all.

Lebert's figures give 3 per cent. as the general frequency of amyloid disease in empyema; others put it higher; but none of these statistics apply to empyema as we meet it now under more favourable circumstances.

Something may depend upon the cause which has produced the suppuration, rather than upon the suppuration itself. Thus it is especially common in syphilis, not infrequent in tubercle, and occurs so frequently in actino-mycosis as to suggest that it is a peculiarity of the disease itself. Amyloid disease is rapidly becoming, I believe, a rare disease in general, owing, no doubt, to improved surgical treatment, and as a complication of empyema it now ceases to be of any practical importance.

ETIOLOGY.—The table on the next page shows the age-distribution, sex and mortality of empyema for twelve years. The figures are taken from the statistical tables of St. Bartholomew's Hospital from the year 1883 to 1894 inclusive.

The number of cases is 254. During the same period 733 cases of serous effusion were admitted into the hospital, so that the relative frequency of serous effusion to empyema is about 3 to 1. Some other statistics show a smaller relative frequency than this, viz., from 14 to 18 per cent., or about 6 to 1.

Sex.—Of these 254 cases, 181 were males and 73 females. This gives roughly the proportion of $2\frac{1}{2}$ to 1, which represents the relative frequency of the disease in the two sexes. This proportion seems to hold throughout the whole of life, and does not vary even in children.

Age-distribution.—It will be seen that there are two periods during which empyema is unusually frequent; first, in early childhood up to the age of 5, and in early adult life between the years 20 and 30.

[1] Hall, *Clin. Soc. Trans.*, 1884, p. 86.
[2] *Cf. D. A. f. kl. Med.*, xxiv. 169. Newton Pitt, *Brit. Med. Jour.*, April 1890.

TABLE showing Age-Distribution, Sex, and Mortality of Empyema for 12 years (1883 to 1894 inclusive).
(Taken from the Statistical Tables of St. Bartholomew's Hospital).

Disease.	Total.	Dischd.		Died.		Under 3.				—10.				—15.				—20.				—30.				—40.				—50.				—60.				Over 60.			
		M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
Age and Sex,	...	143	57	38	16	28	17	10	8	19	15	2	...	11	4	2	1	18	3	2	...	35	11	5	4	15	5	3	1	14	2	9	1	3	...	5	...	...	...	...	1
Totals,		254				63				36				18				23				55				24				26				8				1			
Per-centage,		100				25·0				14·0				7·0				9·0				21·5				9·5				10·2				3·0				...			
Mortality at different age periods; per-centage		in general 21·5, and nearly the same for both sexes.				28·5				6·0				16·0				9·0				17·0				16·0				40·0				62·0				100·0			

In early childhood no less than 25 per cent. of all the cases of empyema occur; and if to these be added the number for the next period of five years, no less than 40 per cent. of all the cases of empyema will be found to occur under the age of 10.

The frequency is at its lowest between 10 and 15 years of age, when it falls to about 7 per cent.; while between 20 and 30 years of age the percentage stands at 21·5.

The side affected.—There is no reason why one side should be affected more than the other, and in 152 cases the sides come out at nearly the same figure, viz., right 80, left 72.

MORTALITY.—The table shows also the mortality in general and at the different age-periods. The general average mortality of empyema is 21·5, and this is practically the same for both sexes.

The mortality, however, varies very much at the different age-periods. Thus, in early childhood, when empyema is so common, it is also most fatal, and at this period 26 per cent. of the cases die.

Between 20 and 30, the mortality is about 16 per cent., but it is at this time that phthisis is most common, and it is well known that empyema occurring in the course of phthisis is more serious than the other forms, so that, excluding phthisical cases, the mortality at this age-period is probably not greater than it is in other quinquennial periods in young people, perhaps about 8 or 9 per cent.

After 40, the mortality increases with very great rapidity. Thus, between 40 and 50, it reaches 40 per cent., and between 50 and 60, it is 62 per cent. These facts are of very great importance in respect of prognosis, though in any given case these general conclusions have to be corrected, and due allowance made for other factors, especially the cause of the effusion.

In considering the mortality I have preferred to take recent statistics, such as those which I have drawn from the records of St. Bartholomew's Hospital, rather than to quote statistics of previous years and other places, because we may assume that all these cases in the last twelve years have been treated in the way now most approved, viz., by early incision and with all the recent antiseptic improvements, so that they represent, on the average, the most favourable results that are likely to be obtained at present; yet in this respect I think every year shows fresh improvement, and the mortality is still, I believe, steadily diminishing.

BACTERIOLOGY OF EMPYEMA.—It is now a generally accepted fact that suppuration does not occur without the presence of pyogenic organisms. As these organisms are of several kinds and differ *inter se* in their virulence and the effects they produce upon the body, it is tempting to enquire whether special organisms can be connected with the different forms of empyema which we have come to recognise clinically. Thus, when empyema follows pneumonia, phthisis, pyæmia, or specific fevers, it would be interesting to know how far the organisms peculiar to these affections are found in the pleura, and whether the organisms are different in those empyemata which arise spontaneously, without any of these causes; whether fœtid empyemata are associated with putrefactive organisms, and whether, again, there is anything bacteriological which can explain the difference observed between the empyema of children and that of adults.

Though these questions have, of recent years, attracted much attention, they cannot be regarded yet as settled; still, so far as investigation goes, the bacteriological results seem to confirm, to a great extent, the results at which clinical observations had arrived.

The organisms found to be associated with empyema are the pneumococcus and the streptococcus, both of which are very common; the tubercle bacillus, which is comparatively infrequent; staphylococcus, the pneumo-bacillus, and Eberth's typhoid bacillus, which are rare. In fœtid empyema numerous common putrefactive organisms are present.

These various organisms are sometimes found alone, and sometimes associated together, but when in association one form is usually predominant.

The following figures are given[1]:—

	Netter		Thue.
Number of cases, ...	110	...	24
Streptococcus	51	...	5
Pneumococcus	32	...	14
Tubercle bacillus	12	...	3
Saprogenic bacilli	15	...	...

Tubercular and fœtid empyemata form about 25 per cent. of all cases; the staphylococcus is found in about 2 per cent. In the remaining 73 per cent. the organisms found are the streptococcus and pneumococcus, which are associated together in about 3 per cent.; but in the great majority of cases, *i.e.*, in 70 per cent., the one or other of them exists alone.

Taking all cases of empyema together, the streptococcus is found in about 44 per cent., and the pneumococcus in about 26 per cent.

But the relative frequency of these two organisms to each other is very different in children and adults, for in the adult the streptococcus is more than three times as frequent as the pneumococcus (53 per cent. to 17 per cent.); while in children the proportions are exactly reversed.

Streptococcus Empyema.—The streptococcus is the organism most commonly found associated with suppuration in the serous membranes of joints and elsewhere, and in the adult, at any rate, it is this organism that is the commonest cause of empyema.

The pleura, in most cases, is not the primary seat of disease, but is involved secondarily from some focus of infection elsewhere. Netter gives 56 cases of streptococcus empyema, of which 12 only could be considered primary, and probably in some of these the source of infection lay in the lung. In the rest the empyema was associated with some source of infection external to the pleura. In many instances (14 out of 44, Netter) this original infection could be traced to some suppurative affection of the peritoneum and pelvis; for example, pelvic peritonitis, subphrenic abscess, abscess in connection with the liver, stomach, or spleen. In others the original suppuration was seated in the mediastinum, in the tissues of the neck or of the chest walls. In other cases, again, the empyema was the result of blood-infection in connection with otitis media, ulcerative endocarditis or general pyæmia; and in some the infection had been introduced from without by careless paracentesis.

But in the majority of instances it is doubtless from the lung itself that the infection is derived, *e.g.*, broncho-pneumonia, phthisical cavities or gangrene of the lung.

To produce empyema the streptococcus must be present in the pleura in considerable numbers; in small numbers it may produce no effect at all, and in moderate numbers a sero-purulent, rather than a purulent, effusion. It is also stated that the fluid may change its character, being at first serous and becoming purulent later when the streptococcus is present in greater numbers; but this is not an absolute rule, for streptococci may be abundant even in serous effusion.

[1] *Bulletin des Hôp.*, 1895, p. 439, quoted from Charcot, *Traité de Médecin.*

The fluid is stated to be very rarely pure pus, but to have a tendency to separate into two layers, the pus forming a grayish, non-coherent sediment, while the supernatant fluid is of a sero-purulent character. It is stated that this sedimentation may be met with in life, though I cannot confirm this from my own observation.

Most of the empyemata which occur in the course of the specific fevers, as well as in the course of pyæmia and fever, are of streptococcal origin, and it is to streptococcal infection that abscess of the brain, secondary to empyema, is stated to be due.

These empyemata vary very much in severity of symptoms. They are sometimes associated with but little constitutional disturbance or fever, and occasionally are completely masked, *i.e.*, found *post-mortem* when there have been no symptoms to draw attention to them during life. This occurs especially in debilitated persons, *e.g.*, patients convalescent from fever.

Netter states that many cases of streptococcus empyema can be cured by paracentesis, but the treatment of empyema by paracentesis will be discussed later.

Pneumococcus Empyema.—In pneumococcus empyema the pus is viscid, greenish in colour, contains much fibrin, and does not separate on standing into layers.

The organisms are short-lived, and are not, as a rule, found after the crisis is past. They live longer in fluid, in the absence of oxygen. They occur occasionally in chains, and may resemble the streptococcus so closely that cultivation and inoculation may be necessary to prove their nature. The organisms often stain badly, and defective staining, it is stated, is an evidence of diminished virulence.

Pneumococcus empyema may be primary, *i.e.*, may develop without any antecedent pneumonia; but the number of these cases is small, and in some of them there may well have been patches of deep-seated pneumonia, which could not be diagnosed. In the majority of cases it follows upon well-marked pneumonia.

In the adult, the streptococcus is three times as common as the pneumococcus, while in the child the relations are exactly reversed. Even in the adult it does not follow that empyema after pneumonia is necessarily due to the pneumococcus; for in the adult, while 30 per cent. of all empyemata follow pneumonia, 17 per cent. only of these are associated with the pneumococcus. It follows, therefore, that nearly one-half of these which are subsequent to pneumonia must be due to other organisms. Empyema is more common in years when pneumonia is frequent; as, for example, when influenza is prevalent; but influenza empyema is very often due to the streptococcus, and not to the pneumococcus.

In the child, again, lobar pneumonia is certainly not a common affection, yet the pneumococcus is found in 53 per cent. of all cases. It is broncho-pneumonia and not lobar pneumonia that commonly precedes empyema in the child.

It would follow, therefore, that broncho-pneumonia in children must often be of pneumococcal origin, and this I have already shown to be the case.

Empyema associated with *Friedländer's bacillus* is rare, but it does not seem to differ materially from that due to the pneumococcus.

Staphylococcus Empyema.—Netter shows that the staphylococcus occurred in 21 cases out of 156, *i.e.*, in about 14 per cent., but in only 2 cases did it exist alone. In all the rest it was associated with other organisms, viz., the pneumococcus in 7, the streptococcus in 4, and the tubercle bacillus in 4.

In most instances the empyema was associated with some other source of staphylococcus infection in the body, *e.g.*, with ulcerative endocarditis, or pyæmia, and in one or two cases the infection was evidently introduced into the pleura by the needles used for exploration.

Eberth's Typhoid Bacillus has been occasionally found in empyema occurring in the course of typhoid fever, but these cases, as in other specific fevers, are associated more frequently with the streptococcus or staphylococcus.

Tubercular Empyema.—Empyema occurring in the course of phthisis is very often due to the streptococcus or pneumococcus, and is not associated with the tubercle bacillus in more than 10 per cent. In 13 cases Netter found the tubercle bacillus in 5, the staphylococcus in 4, non-pathogenic organisms in 3, and none at all in 1.

The bacilli are often as difficult to find by the microscope as they are in serous effusion, yet inoculation and cultivation are more successful. Thus Netter succeeded by cultivation and inoculation in 12 out of 13 cases, a proportion of positive results much greater than with serous effusion. Occasionally the fluid is sterile; if an empyema be sterile, or contain staphylococci only, the presumption is strongly in favour of a tubercular origin.

It is asserted that in tubercular empyema the parietal layer is more affected than the visceral, suggesting that the disease is secondary to some tubercular affection of the ribs or mediastinal glands rather than to tubercular disease of the lung. The pleura is much thickened, rough, with caseous masses, in which are found characteristic tubercles and tubercle bacilli. The fluid, too, is often sero-purulent, contains no fibrinous flocculi, and often comparatively few pus cells, and deposits a powdery sediment, in which, besides much granular detritus, fat drops and fatty crystals may be found.

These empyemata being of insidious origin, slow development, and unattended with serious general symptoms, may run a long course and the effusion reach a large size.

Empyema in the course of phthisis, no matter what organism it be associated with, is of grave prognosis, and in the end usually results in death.

FORMS OF EMPYEMA AND THEIR DIAGNOSIS.—The general diagnosis of pleuritic effusion, the conditions with which it is liable to be confused, and the means of distinguishing between serous and purulent effusions, have been already dealt with. Here we have to consider certain special forms of empyema and the difficulties which they are likely to cause in diagnosis.

Empyema may be divided into two main groups, viz., *general* and *localised*, according as the whole pleural cavity is affected or part of it only. Besides these, there is a form which is called "*loculated*," *i.e.*, a pleural effusion in which the pleura is divided into compartments separated from each other, each possibly containing different kinds of fluid.

Loculated Empyema.

Loculated polymorphic empyema is a curiosity, and a comparatively rare one, more often discovered after death than diagnosed during life, though occasionally exploration with the needle may determine its nature by withdrawing different kinds of fluid by puncture from different parts of the chest.

In such a case, we shall have to distinguish between loculated empyema in which two or more different compartments exist in the pleura, and the condition already referred to, where, by subsidence, the lower strata of the fluid are markedly purulent and the upper only turbid. This condition, as

already stated, is very rare, and if doubt existed, disturbance of the patient would soon settle the diagnosis by causing the supernatant fluid to become more purulent and turbid.

Jaccoud's [1] statement, that these cases may be diagnosed by the presence of vocal fremitus along the lines of attachment of the lung to the thoracic walls, is not supported by much evidence in his communication, and I do not think the statement is in accord with facts.

Instances of polymorphic pleurisy.—I tapped a man of 54 and removed a large amount of serous fluid. He did not get well until he brought up several ounces of fœtid pus. In this case the fœtid empyema was the initial lesion, and this gave rise to the serous effusion, which became predominant and was tapped.

The most remarkable instance I have met with of loculated empyema occurred in a boy with general pyæmia. An incision was made in the left side in the fifth space in the axilla, and pus was evacuated. The pericardium was found bulging, and was incised also through the pleural wound. It proved to contain pus too. When the lad died, two other compartments in the pleura were discovered, one above and the other below the incision. These had not been opened. The lower contained turbid sero-purulent fluid and the upper serous fluid only.

Another very remarkable case is recorded by Pel,[2] in which an empyema on the left side was laid open in the usual way, and shortly after considerable bulging of the pericardium was observed which was thought to be due to pericardial effusion. This was opened and 2 litres of pus evacuated (3½ pints). On *post-mortem* examination the pericardium was found to be universally adherent and greatly thickened, and in the cavity in the pleura which had been laid open was another collection of pus anterior to that first opened, and separated from it by firm adhesions in the anterior axillary region.

Cullingworth [3] also records a case in which serous fluid was removed from the pleura, and on the patient's death an empyema situated between the diaphragm and base of the lung was found to which the serous effusion was obviously secondary. The existence of this had not been suspected.

Another curious case is recorded by Gaillard.[4] One compartment contained pus; this was in front; another, behind, contained serum. The autopsy showed that the pleural cavity was divided into two by a vertical fibrous septum.

Localised Empyema.

Empyemata of this kind are the most difficult of all to diagnose, and are frequently overlooked. Even if the presence of pus be suspected and a needle introduced for exploration, it may require some skill, and courage too, to find it.

In one case of this kind it was not until the needle had been thrust about 6 inches into the side, and directed from the axilla to the middle of the vault of the diaphragm on the right side, that pus was found. The needle was used as a director, and an incision made through dense tissue. Even after a free incision the tract was 4 inches long before the cavity was reached by the drainage tube. The patient made complete recovery.

The two common forms are *diaphragmatic* and *basic*, but localised empyemata may occur *in other places*, and it has already been laid down as a general rule that a localised effusion in a peculiar place is presumably purulent. Some of these may have originated in interlobar suppurations, *i.e.*, between the lobes of the lung, *interlobar empyema*; others in connection with other foci of inflammation or disease in or near the thorax.

A man of about 35 years of age presented the signs of an empyema in the mid-axillary region. It was correctly located, and opened. It proved to be quite superficial and shallow, but did not heal. The man died of other causes some time later. The autopsy showed the empyema cavity to have been funnel-shaped, with a long and narrow neck formed by a fistulous track several inches long and not larger than a pencil, extending back to one of the dorsal vertebræ, which was carious.

[1] *Acad. de Méd.*, 1879.
[2] *Berl. klin. Woch.*, 1884, No. 8.
[3] *Brit. Med. Jour.*, 1886, ii. 17.
[4] *Soc. méd. d'Hop. de Paris*, Nov. 24, 1895.

In the case of diaphragmatic and basic empyema, the diagnosis will have to be made from suppuration below the diaphragm, *e.g.*, on the right side from subphrenic abscess and abscess in the liver, and on the left side from suppurative peritonitis in connection with ulcer of the stomach, etc.

When the mischief is on the *right side* and below the diaphragm—such as, for instance, subphrenic abscess, abscess or hydatid in the liver, and some cases of new-growth—the diaphragm is thrust upwards into the thorax, so that the line of dulness obtained on percussion is markedly curved, rising to its highest in the nipple line or axilla, and falling both towards the back and towards the front.

If the disease be in the right lobe of the liver, there is some enlargement of that lobe downwards also; it is not, as a rule, difficult to distinguish between these affections of the liver and pleuritic effusion.

In the case of subphrenic abscess the difficulties are greater, for not only is the diaphragm thrust up, but the liver thrust down; in other cases the suppuration is not so circumscribed, but spreads itself more uniformly over the whole surface of the liver, so that the displacement of the diaphragm upward is not so marked.

On the left side, the diagnosis is more difficult, for there is not a solid organ like the liver, which will give the abscess purchase from below and enable it to thrust the diaphragm upwards to the same extent. In both cases alike, when the suppuration is near the diaphragm, but especially on the left side, if pain be felt, it may be referred to parts either above or below the diaphragm indiscriminately. Thus, in the case of the pleura, it may be referred to the abdomen, and with a localised peritonitis it may be referred to the chest.

For example, in a case of localised suppurative peritonitis in connection with gastric ulcer, which ultimately caused the patient's death, the symptoms, though at first referred to the region of the stomach, afterwards were entirely referred to the pleura, so that the case was at one time thought to be an empyema, and an exploratory puncture was made. No pus was found, and no operation on the pleura therefore performed. Even up to the time of the patient's death there were no abdominal symptoms complained of, although, *post-mortem*, the patient was found to have a general suppurative peritonitis, which had commenced round a gastric ulcer.

In the case of empyema in peculiar places, the diagnosis will depend upon the site. When near the heart it would have to be distinguished from pericardial effusion, from some affection of the heart, or from an aneurysm—diagnoses which might become extremely difficult if the empyema pulsated. Higher up, it might be confused with suppuration or some other affection in the mediastinum or in the lung, or even with thoracic aneurysm or new-growth.

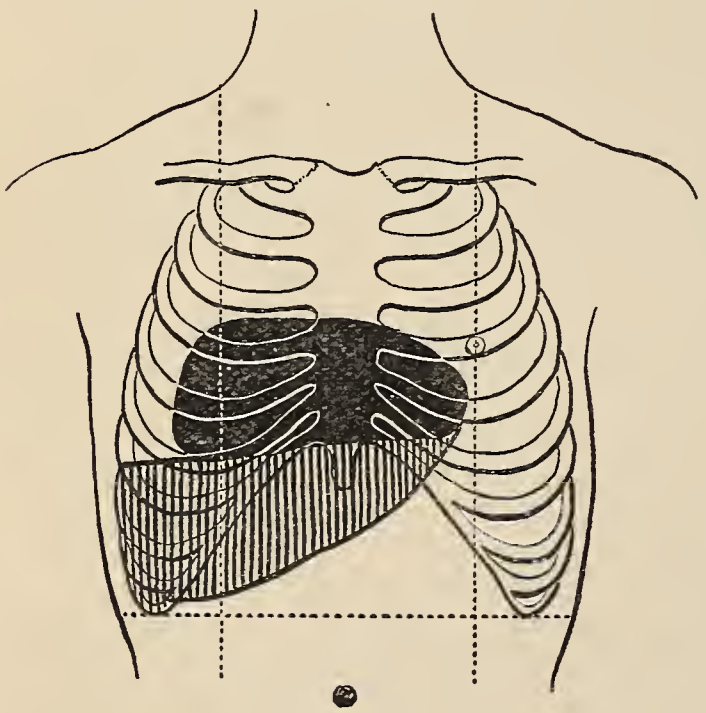


Fig. 151.

The following case is a very rare one, in which a localised empyema developed in immediate relation with the pericardium, as the figure shows. The diagnosis was extremely difficult, but it was successfully made, though it was not until the incision was made and the pus evacuated, so that the finger could be introduced and the region explored, that it was conclusively proved that the abscess was not in the pericardium, but outside it. Although in immediate relation with the pericardium, this abscess did not pulsate externally.

Chronic abscess of the chest wall, again, may cause difficulty in diagnosis, for it may not be easily distinguished from a pointing empyema. This has been

called *peripleuritic abscess.* As a rule, when an empyema is pointing, the superficial inflammation is more acute; there is more heat, tenderness, and redness of the skin than is usual with most of these chronic abscesses; over and above which, in many of these cases there are no physical signs indicating mischief in the lung or in the pleura.

Such abscesses are, for the most part, tubercular in origin, and when incised run a very chronic course, like a tubercular abscess elsewhere. They may, of course, be connected with a carious rib, but a large number of them rise spontaneously in the subcutaneous tissue, possibly in connection with a lymphatic gland.

When associated with physical signs in the chest they may present greater difficulties in diagnosis. In phthisis they are not altogether uncommon, but the diagnosis can generally be made by the fact that the dulness does not extend far beyond the abscess, as would be the case if there were an empyema present, even though it were a localised one. The common seat of these abscesses is in the mid-axillary or anterior-axillary region, but they may be found in almost any part of the chest, though the upper part is the rarest of all.

When close to the heart they may pulsate, and then the diagnosis becomes extremely difficult.

I saw a case, not long ago, in which there was a tumour close to the apex of the heart, which pulsated so distinctly that no one ventured to interfere with it, thinking it to be an aneurysm. Ultimately the skin gave way and a discharge of pus took place, when the tumour disappeared.

Recurrent Empyema.

Some of the most difficult cases to diagnose are those in which a second empyema occurs upon the same side as had been previously affected. These are called recurrent empyemata, and there may be an interval of some years between the attacks, even as long as twenty-two years in a case referred to by Wilson Fox.[1]

Cases of recurrent empyema are extremely rare. I recorded one instance of this kind a little while ago. It occurred in a boy of 15 years of age, who presented the signs of fluid in the left side of his chest, the heart being displaced to the right and the dulness extending across the sternum. The signs pointed to the presence of fluid, a diagnosis which would have been simple enough if the patient had not given a history of an empyema eight years previously, for which he had been operated on in the Evelina Hospital, the scar of the operation being visible in the posterior-axillary line. However, a needle was inserted, and after two unsuccessful attempts pus was discovered and a free incision made. The temperature immediately fell to normal, and the patient got well. The patient did not appear to be tubercular, and no cause for the second empyema could be found.

An analogous case was met with in a man of 25, who came under observation for an empyema of the right side. He had had the same side tapped for serous effusion thirteen months previously, with the removal of 30 ounces of clear serous fluid. The side was opened, 130 ounces of pus removed, and a portion of rib excised. As the cavity did not close, six months later an inch of the fourth, fifth, sixth, and seventh ribs were removed, and three months later the cavity had almost closed except for the tract of the tube.

Another case of the same kind occurred in a man of 29. He, too, had had serous effusion twelve months previously on the same side; this case, however, got well with an ordinary incision, only without excision of rib.

In some cases the interval between the recurrence of the empyema is short, a few weeks or months it may be. These may be called *recrudescent* or *relapsing* rather than recurrent. They are probably really of the nature of residual abscesses which have taken on fresh action.

[1] P. 1082.

Empyema with Malignant Disease.

Malignant disease of the lung and pleura may, of course, be associated with effusion, but this is usually serous and often more or less blood-stained, while the temperature is normal. Purulent effusion is rare.

Speaking generally, the diagnosis is not difficult; but there is one form in which the difficulties may be considerable. I refer to that in which the malignant growth has developed round the root of the lung, compressing the bronchi and vessels, and thus leading to the condition which has been described as "solid œdema of the lung." In this a sub-acute form of inflammation may occur, giving a rise of temperature which may strongly resemble the hectic of phthisis or of empyema.

As the affected part is dull and yields no vocal vibrations or vocal resonance, the existence of pleuritic effusion will be suspected, and from the temperature it will be thought to be purulent. The result of exploratory puncture is generally negative.

In one case of this kind exploratory puncture in the middle region of the axilla obtained pus, and a small incision was made, with the result of evacuating a drachm or two of a purulent fluid. The diagnosis of malignant disease of the lung had been previously made.

On the *post-mortem* examination the cavity from which the puriform fluid was obtained proved to be in the lung, and to be due to a necrotic, disintegrating change, produced, no doubt, by obstruction to the circulation, for the patient had malignant disease surrounding the root of his lung, and compressing both the air-tubes and blood vessels.

The difficulty of diagnosis from some forms of pneumonia, in which the tubes are plugged with secretion, has already been referred to. In these cases the absence of vocal vibrations and vocal resonance suggests the presence of fluid; but as a rule, even if the physical signs be conclusive, the general aspect and course of the case make the diagnosis clear; for with empyema the symptoms are not, as a rule, so severe, nor the onset so sudden, and of course the duration and course of the two diseases are different; the only difficulty which then arises is in those cases in which the empyema follows the pneumonia.

Pulsating Empyema (*Empyema pulsans*).

Empyemata may pulsate, but distinct pulsation is certainly a very rare phenomenon.

When Comby [1] wrote his paper, in 1882, he did not succeed in finding more than about 30 cases recorded. Wilson,[2] in 1893, brings up the number of published cases to 68, but the increase in numbers of recent years is chiefly due to the larger number of cases recorded in which there has been a general shock, more or less distinct, felt over a considerable area of the side. The fact remains that distinct pulsation is very rare, but the condition is one which always attracts attention, on account of the difficulties of diagnosis to which it may give rise.

The pulsation is of two kinds—

1. A general shock or impulse communicated to a considerable area of the chest walls. This has been called *intra-pleural pulsation.*
2. Expansile pulsation localised in an external tumour. This has been called *extra-pleural pulsation*: it is sometimes visible, but often only to be felt by the hand. It occurs, with but few exceptions, in the left side. Thus of 66 cases, it was on the right side in 5 instances only.

[1] *Arch. gén. de Méd.*, Nov. 1883. Thesis, *L'Empyème pulsatile.*
[2] *Trans. Assoc. Amer. Phys.*, Philad., 1893, viii. 195.

It is synchronous with the heart, and almost invariably systolic in time, though there are 3 cases in which it has been stated to be diastolic, 2 recorded by Macdonald [1] and 1 by Stokes.[2]

The condition may be met with in either sex and at any age. It is more common in males, in the proportion of about 4 to 1, and in adult life between the ages of 20 and 35 ; the youngest recorded case was 2 years of age, and the oldest 57.

It is rare except with empyemata, and then only with those of long duration ; but it is described also with serous effusion and with pyo-pneumothorax.

Intra-pleural pulsation.—When the pulsation is intra-pleural it is occasionally visible, but more often only to be felt by the hand. Usually it is widespread and felt over the lower part of the chest, especially in the axilla, as the patient is lying in bed. Occasionally it is felt higher up and more in front. It may be limited to one or two intercostal spaces in the supra-mammary region, or be more diffuse and extend outwards from the sternum to the middle or posterior axillary line, and downwards from the third rib to the lower part of the axilla. In these cases, especially where the pulsation is felt high up, it is probably analogous to that which is felt in the abdomen in ascites, and partakes of the character of a fluid-wave.

Intra-pleural pulsation may occur with any kind of effusion ; chiefly, it is true, with empyema, but also with pyo-pneumothorax, and sometimes even with serous effusion. Of the last, Traube [3] recorded the first case, but several others have been described in recent years.[4]

Extra-pleural pulsation, that is, pulsation limited to an external tumour, is met with in empyema only. It occurs when the empyema is pointing, and if the empyema be pointing in more than one place, may occur in two or three of the tumours.

Thus, of the cases recorded,[5] in 3 instances there were two, and in 2 instances three, external tumours, all of which pulsated.

In many of these cases the tumour and the pulsation in it may vary with respiration, the tumour becoming larger and the pulsation more distinct with expiration, and less so with inspiration ; sometimes both the tumour and the pulsation in it may be made to disappear by pressure, that is to say, the pus may be returned within the thorax.

These pulsating tumours may be met with in any part of the chest where an empyema points, but most of them somewhere near the heart, and of course this will be their most likely position if the empyema be localised. Thus they may occur above the præcordial region, but are found more commonly in the mammary region or near the apex, and occasionally even immediately over the præcordium in front.

Where there is more than one tumour, they are usually close together, but they may be widely separated, *e.g.*, one tumour may be near the heart and the other close to the spine (Macdonald, Wilson Fox).[6]

Occasionally, if the empyema has tracked, a pulsating tumour may be found in a very unusual place, for example, in the loin.

[1] *Dubl. Jour.*, 1854, xxv.
[2] *Dis. of Heart and Aorta.*
[3] *Ges. Beitr.*, 1871.
[4] Fraentzel, *Ziemssen*, iv. 2, p. 355.
[5] Wilson, *l.c.*
[6] *Dubl. Jour.*, 1854, *l.c.*

Thus Owen Rees[1] records a case in which the pus tracked to this spot, and the *post-mortem* showed that it was due to an empyema; the pulsation in this case being transmitted, not from the heart, but from the abdominal aorta, with which the abscess was in close relation. A similar case is recorded by Müller.[2]

Diagnosis.—The diagnosis may be very difficult, but the difficulties are not quite the same in intra- and extra-pleural pulsation.

Intra-pleural pulsation does not, as a rule, present any great difficulties, for it is rarely met with except with large effusions, and then the nature of the case is evident. If it should be limited, however, to the anterior part of the chest, or to the region of the heart, it might need to be diagnosed from a dilated or hypertrophied heart on the one hand, or from an aneurysm of the heart or of the aorta on the other. It is not, however, in these cases that the difficulties occur, or if they do, that they are of any great practical importance.

The association of the two conditions, namely, of a pulsating pleural effusion, with an aneurysm of the aorta, is very rare. I only know of one instance, that recorded by Stokes,[3] and in that case the aneurysm was very small.

The diagnosis from pulsating pneumonia and a pulsating intra-thoracic new growth, as suggested by Graves, is very unlikely to cause difficulty; for these cases are, to say the least, extremely rare. I have never seen a marked instance of either. And as to the very existence of pneumonia-pulsans, I am very sceptical.

With extra-pleural pulsation, that is, pulsation in an external tumour, the difficulties are also not as a rule great, for the physical signs indicate the existence of a large empyema which is pointing.

If the empyema be localised, especially in the neighbourhood of the heart, the difficulties of diagnosis may be greater.

If the tumour be near the apex of the heart, or over the lower part of the præcordial region, the question of cardiac aneurysm may arise; yet aneurysms of the heart hardly ever cause an external tumour in this position. They develop rather in other directions within the thorax.

I have, it is true, seen one case of cardiac aneurysm causing an external swelling. This occurred on the right side of the chest, between the nipple line and the lower part of the sternum; but there was no doubt about the diagnosis.

Where the swelling is higher up, and especially if it be in the first and second intercostal space, the chances of aneurysm will be greater.

Thus, in Topham's[4] case, in which there was a pulsating swelling in the third intercostal space, close to the sternum, an aneurysm was diagnosed, and the patient was kept in bed for about three years. A discharge of pus then took place, the tumour disappeared, and the patient got well.

In Aran's[5] case an abscess in the upper part of the mediastinum caused an external swelling. This was not connected with the pleura; it discharged, and the patient recovered. In Vidal's[6] case, a similar pulsating swelling over the lower part of the sternum proved also to be due to a small abscess in this position; this patient was suffering from double empyema, though no direct communication could be traced between the mediastinal abscess and the pleura.

Localised empyemata in the neighbourhood of the heart are very rare, and even when they occur do not often cause external pulsation. In the case I have already quoted, though the empyema was in close apposition with the right side of the heart, there was no trace of pulsation communicated externally.

[1] *Brit. Med. Jour.*,1858, Aug. 21.
[2] *Berl. klin. Woch.*, 1872, Jan. 22.
[3] *Dis. of Heart and Aorta.*
[4] *Lancet*, 1878, i. 756.
[5] *Bull. Soc. Méd. des hôpitaux*, xv. 96, 1858.
[6] *Bull. de Soc. Anat.*, 1854, p. 243.

On the other hand, even when the tumour is due to an aneurysm, it need not necessarily pulsate, for the interior may be filled with clot.

The difficulties of diagnosis are greater between a local abscess of the chest wall, which pulsates, and an aneurysm, than between an aneurysm and an empyema, because in the majority of cases the signs of an empyema are so characteristic.

Superficial abscesses are most likely to cause difficulty when they are seated somewhere near the apex of the heart, where the pulsation is forcible and the intercostal spaces fairly large, and they are still more likely to pulsate if there be pus beneath the ribs as well as above them; as, for instance, with a periosteal abscess, due to caries of a portion of the rib in this position.

I have seen one instance of this kind in which the pulsation was so marked that no one cared to interfere with the tumour. Ultimately the skin burst, pus was spontaneously discharged, and the tumour entirely disappeared.

I think, in cases of this kind, a small puncture with a very fine needle, for the purposes of exploration, would do no harm whatever, even if the tumour proved to be an aneurysm; and of course it would at once clear up the diagnosis if pus were obtained. Over such a tumour, a murmur would not, as a rule, be present, as it might be in the case of an aneurysm; but then a murmur is not necessarily present even if the tumour be aneurysmal, and, as stated, some of these external aneurysms do not even pulsate.

Pulsation being, as a rule, absent in effusions of all kinds, of whatever size they may be, it is evident that its occurrence requires peculiar conditions. There appear to be three different kinds of pulsation, and the same explanation will not fit them all.

(i) There is, first of all, the general shock felt over the lower part of the chest, usually in the posterior axilla and behind. This is found only with very large effusions, and necessitates considerable intra-pleural pressure. It is the same thing as the general shock which is transmitted sometimes to the chest walls from the heart through a solid mediastinal or pulmonary tumour, or through a massive pneumonia.

(ii) There is, next, that form of pulsation which is met with chiefly in front and in the upper part or mammary region of the chest, and which does not, as a rule, extend beyond the anterior or mid-axillary line. It is to this group that most of the recently-recorded cases appear to belong. This kind of pulsation is, I think, strictly analogous to that which we meet with in the abdomen in ascites, and is due to the propagation of a fluid wave from the heart to the surface of the fluid. It is felt where the surface of the fluid would be, as the patient is lying in bed upon the back, and would, I think, very likely alter its position if the patient sat up, though I do not know that this has been actually described. As in the abdomen, it is to be connected with a certain laxity of the chest walls, for fluctuation is not nearly as distinctly felt in the abdomen when the walls are tense as when they are relaxed. For this reason this form of pulsation occurs usually with empyemata, and generally with empyemata of some duration; though it might possibly be met with in other forms of effusion, and also with pneumothorax.

(iii) In the third form of pulsation, namely, that in which the pulsation has more of an expansile character, whether it be felt over a large portion of the chest, or limited to an external tumour, the explanation is, I think, different.

This last kind of pulsation is more like that which we get in a large abscess, and which we speak of as a "surgical fluctuation"; and in order that this should be obtained, the fluid must be contained within some more or less resistant walls, that is, must be under some pressure.

Wherever there is a large collection of fluid in the pleura, on the left side especially, it is obvious that the pulsations of the heart must affect it; but in most cases the pulsations spend themselves upon the least resistant part of the abscess walls, namely, upon the incompletely collapsed portion of the lung or upon the mediastinum, or, if pneumothorax be present, they exhaust themselves in compressing the air in the pleura.

In order that the pulsation should be transmitted distinctly to the chest walls, or to an external tumour, it is necessary that the other parts of the walls of the pleura should be rigid and resistant, so as to enable the pulsations to be transmitted to the thoracic walls, *i.e.*, to what are usually the more resistant parts. In the case of a general empyema, *i.e.*, of an empyema occupying the whole pleural cavity, therefore, this kind of pulsation pre-supposes the rigid fixation of the internal walls, namely, of the heart and mediastinum, and of the lung also. This is exactly the condition which has been on many occasions described.

Of this, Comby's case is a good instance. On *post-mortem* examination the heart was found to be rigidly fixed to the right side of the chest, and the lung to be adherent all along the mediastinum, from the sternum to the spine, as well as at the apex, and to be in a condition of extreme fibroid induration. The empyema in this case was a large one, and the pulsation distinct; 80 ounces of pus were removed and the pulsation disappeared, until the fluid reaccumulated, when it returned.

For this kind of pulsation, therefore, we require that the internal walls of the pleura, the mediastinum and lung, should offer almost as much resistance to the pulsation of the heart as the chest walls, and such conditions are not likely to be common.

Where the empyema is pointing, that is to say, where the pus has made its way through the chest walls, and there is a fairly free communication with the pleura, the resistance is very much less; and we should expect, *a priori*, that pulsation would occur in these conditions much more frequently. This we know to be the case, for this kind of expansile pulsation is met with most frequently with external tumours, that is to say, where the empyema is pointing.

In the same way, where the empyema is localised, pulsation of this kind necessitates considerable induration of the walls surrounding the abscess, and probably a considerable amount of intra-pleural pressure.

These cases are interesting chiefly on account of the difficulties of diagnosis, but the diagnosis once made, the treatment is that of empyema, *i.e.*, they may be tapped or opened as may seem desirable, and with success.

It has been stated that pulsating empyemata are, almost without exception, fatal. This is an over-statement of fact, and is far from true. Thus in Wilson's cases, out of 46 in which the result is stated, 24 recovered, *i.e.*, about 52 per cent. The cases which are generally fatal belong to the last group which has just been described, viz., that in which there is expansile pulsation of a very marked kind. These cases are fatal, not because the empyema pulsates, but because of the peculiar conditions, viz., the fixation of the heart, and the changes in the mediastinum and lung, which prevent cure. Of the other cases the great majority recover.

Double Empyema.

Empyema is sometimes double, and it is important to ascertain whether these cases require any different method of treatment.

The rule used to be laid down that with double empyema one side might be opened in the usual way, and that there was no choice but to treat the other

by paracentesis; because, it was argued, incision on the one side having already caused the collapse of one lung, incision on the other would lead to collapse of the other lung and a fatal result.

No doubt, to open the general cavity of the pleura in a healthy man would be a serious matter, but the general rule above stated does not take into consideration the fact that, in the majority of the cases of double empyema, the empyema on both sides, or at least on one, is encysted, that is, localised or limited by adhesions which have formed round it, so that incision would not lay the general pleural cavity open.

Although this is, no doubt, generally speaking, the case, it is not necessary to go so far as some writers, and say that there is no difference between a general and a localised empyema, inasmuch as they are all more or less limited by adhesions. No doubt this is generally the case, but still there is all the difference in the world between the large and the small collections of pus in the pleura.

Theory must yield to practice in these matters, and experience shows that, in cases of double empyema, both sides may often be treated in the same way by free incision, with perfect safety and great benefit.

Two papers in the Clinical Society's Transactions, viz., one by Coupland and Gould,[1] and another by Carr,[2] giving accounts of their own cases, and a list of others, bring the literature fairly well up to date. With the cases there recorded, and a few others which have been added since, I have found a series of fifteen, in all of which both sides were opened and recovery took place. They were all in children, the oldest being 10 and 14 years of age respectively.

The case upon which the first-mentioned paper is founded is perhaps one of the most interesting, because an attempt was first made to cure the double empyema by paracentesis; the objection to free incision on both sides being based on the theoretical grounds referred to.

This child, of the age of 7, owed the empyema to an attack of pneumonia, about six weeks before she came under observation. In the course of five months the left side was tapped eight times and the right four times. The right side was then opened, a portion of rib excised, the pleura washed out, and about 10 ounces of thick pus removed. Nine days later the left pleura was opened and treated in the same way, and 18 ounces of pus removed.

This was followed by complete cure in a few weeks' time, and the child was left with little or no thoracic deformity.

Although in the case just recited paracentesis failed, still it is sometimes sufficient to effect cure, as in the case recorded by Broadbent and Cheatle,[3] in which the right pleura was tapped twice and the left once, and the patient made a complete recovery; in another recorded by Sangster, in which, after the right pleura had been opened with the evacuation of a considerable amount of sero-purulent fluid, the left was successfully treated by paracentesis, the side being tapped twice, with the removal of 2 ounces and 6 ounces of pus respectively.

In the majority of the instances recorded the empyema was of some duration, and was, no doubt, localised; but these cases, at any rate, show that there is no reason why, in a double empyema, both sides of the chest should not be treated in the same way.

It is generally thought desirable to leave an interval between the two operations, and, if necessary, after opening the one side, to gain time by performing paracentesis upon the other. In most of the cases about a week was left between the two operations, sometimes a little longer; but in one of them the second side was successfully operated on only two days after the first.

Fox,[4] in a girl of 12 years of age, had both sides opened at the same time; no urgent symptoms developed, and recovery was complete.

[1] Vol. xxiv.
[2] Vol. xxvi.
[3] Sutherland, *Lancet*, 1894, June 9, collates 21 cases.
[4] *Lancet*, 1894, i. 384.

A more remarkable case is recorded by Cooke,[1] in a baby 17 months old. Both sides were opened at the same time, and 14 to 15 ounces of pus removed from each. The operation caused no urgent symptoms, and recovery was rapid and complete.

Fœtid Empyema.

In fœtid empyema the fluid has an extremely fœtid, sickening odour, such as is met with in gangrene of the lung or in fæcal abscess; it is dirty green or brown in colour, opalescent, turbid, with a pulverulent sediment, and contains more or less altered blood. Microscopically, there are found many red blood cells, pigment granules, fatty crystals or plates of cholesterin. Micro-organisms are numerous and of many kinds, but none characteristic.

The cases of fœtid empyema fall into *two groups*. In the one it is consecutive to putrefactive or gangrenous inflammation in the neighbourhood of the pleura, and in the other it is of spontaneous or primary origin.

Group 1.—When there is gangrenous or putrefactive inflammation near the pleura, the consecutive empyema may be fœtid, though not necessarily so. Thus it is the rule with gangrene of the lung, but rare with other lesions of the lung, even with fœtid, bronchiectatic, or other cavities; if it occur with a pneumonia, the pneumonia must be of a septic and not a simple character.

It may also follow caries or necrosis of the bones of the thorax, but usually the resulting empyema is not fœtid.

It is especially likely to occur where there is a perforating disease of the digestive track, as in connection with stricture of the œsophagus, ulcer of the stomach, or with any fæcal abdominal abscess which has reached the neighbourhood of the diaphragm; or where there are lesions in the lung or liver which are likely to become gangrenous, *e.g.*, a suppurating hydatid cyst.

In this group the symptoms, prognosis, and treatment depend chiefly upon primary disease. The symptoms are asthenic in type. The patient is in a state of profound prostration, with low, muttering delirium, a feeble pulse and dry tongue, and passes into the typhoid state. If the primary disease cannot be relieved as well as the empyema, death must be the result.

Group 2.—In the second group, that is to say, where the fœtid empyema is primary, *i.e.*, of spontaneous origin, and not connected with any other putrefactive lesion, the conditions are entirely different.

It seems that nearly one-half of all the cases of fœtid empyema belong to this group.

Of 20 cases which Netter examined, 7 were due to gangrene of the lung, 5 to pyæmia, and 8 could be attributed to no cause at all, that is, they belonged to the primary group. Schwartz's results are much the same: of 15 cases, 9 belonged to the primary group and 6 to the secondary.

In most of these cases the pus is fœtid, however early in the attack it may be examined, so that it has probably been fœtid from the commencement. It is true that sometimes a simple empyema may become fœtid after operation, but this is quite a rare event, and, as a rule, it is the exact contrary which occurs, for as soon as the fœtid empyema has been opened the fœtor passes off and the pus becomes sweet.

I do not know that there is anything in the symptoms of the case by which the fœtid nature of the effusion may be diagnosed beforehand.

I am inclined to think that in this group the empyemata are generally localised; at any rate this has been so with all the cases that I have seen.

[1] *Lancet*, Oct. 2, 1898.

The prognosis seems to be neither better nor worse than that of ordinary empyemata.

The treatment is the same, except that as soon as the empyema is known to be fœtid, the sooner an operation is performed and the fœtid pus removed the better; and it appears to me desirable, at the same time, to thoroughly wash the side out, and, if necessary, to repeat the irrigation as often as the fœtor returns. A single washing out is often sufficient, and the fœtor may then pass off very rapidly even in a day or two.

I have seen several of these empyemata burst through the lung, and then the diagnosis from a gangrenous cavity in the lung becomes very difficult, if not impossible. The expectoration of a fœtid empyema is, however, no more likely to cause secondary changes in the lung than a simple empyema, and in most of the cases that I can recall, complete recovery has taken place without any complication.

I have seen unusual complications follow fœtid empyema once or twice. Thus I remember the case of a young lady who had an empyema, the fœtid nature of which was not known until the side was opened, when the stench was so overpowering as to turn the surgeon sick. The case, however, ran its ordinary course, except that shortly after operation the patient developed a severe diphtheritic inflammation of the conjunctiva which ultimately destroyed the cornea and necessitated the excision of the eye. In other respects the case ran the ordinary course and ended in complete recovery.

THE TREATMENT OF EMPYEMA.—When pus is known to exist in the pleura, the only active course open is to remove it by a surgical operation.

If the pus be left alone, that is, if nothing be done—for no drug is known which will check the formation of pus or cause its absorption—it is, of course, theoretically possible that the formation may cease, and that what has formed may inspissate and become encapsulated, or possibly may disintegrate and become partially or even completely absorbed. This, however, can only occur where the amount of pus is quite small, and is not at all likely to happen even then. In all other cases the pus continues to increase, and in the end makes its own way out of the pleura; usually, either through the lung or the chest walls, and occasionally in other less desirable directions. In the latter case it will lead to complications which may prove grave or even fatal. Even in the former more favourable directions, cure can only occur after long illness and much suffering.

To leave the pus, therefore, to itself is bad practice if anything else can be done; in other words, pus having been diagnosed, it should be removed. This can be effected either by paracentesis or by free incision.

The choice of operation can be made at leisure; for, even if the effusion be large and the symptoms urgent, paracentesis will be sufficient to give immediate relief, and thus time will be gained for the full consideration of the best method of procedure. Even if free incision has already been decided on, a preliminary paracentesis may be of use, for in hospital practice, patients often come under observation very much neglected and run down, and by paracentesis time will be gained, during which the patient may recover strength, and be brought into a better general condition for operation. In operations on the chest, as in other surgical operations, success, to some extent, will depend upon the choice of the right time for operation. Although there should be no unnecessary delay, still there need be no undue hurry; and in choosing the time for operation clinical experience will greatly aid the judgment.

Paracentesis, repeated if necessary, suffices sometimes to cure empyemata, and in some cases this may be the wisest and safest method of treatment. In most instances, however, whether paracentesis has been previously performed or

not, free incision ultimately becomes necessary ; and, as a rule, this forms the most expeditious and simplest mode of cure. That being so, it will be well to consider the treatment by free incision first, and to reserve paracentesis for subsequent discussion.

Free Incision.

In all cases, before making any incision, it is important to be certain that pus is present at the spot where the incision is to be made, and this can only be ascertained by means of an *exploratory puncture* with the needle. This should on no account be ever omitted, for although nothing is easier than to find the pus in an ordinary case of empyema, still, in other cases, the difficulties may be very great, and it is never possible to tell beforehand, for certain, whether the case is simple or difficult.

Thus I remember the case of a child in which the evidence of a large empyema was quite conclusive, and in which the facts seemed so clear, that I was on the point of waiving the preliminary puncture, when the house physician reminded me that I was breaking the rule that I was constantly preaching, and I determined to insert the needle. Simple as the case appeared, it was not till after two or three punctures that I succeeded in finding pus, and then, of course, the incision was a simple matter. Examination of the pleural cavity with the finger showed that the empyema was a very peculiar loculated one, and the difficulty experienced with the puncture was explained.

In localised empyemata, also, the difficulties may be great.

I remember an adult who had a very considerable area of dulness at the base, with other signs of suppuration, and there could be no doubt that pus was present. Yet I made exploration with the needle on no less than eight separate occasions without finding where the pus was, and it was not till the ninth occasion, when I gave an anæsthetic and put the needle three or four times into different parts of the chest, that at last I succeeded in reaching pus. Nine ounces were removed, and this sufficed to cure the patient.

In another case the pus was seated in a pouch between the diaphragm and base of the lung. There was no means of ascertaining that fact by physical signs, and though the case seemed simple, very great difficulty was experienced in finding the pus, and it was not until the needle had been thrust more than 4 inches into the chest that pus was obtained. The needle was then used as a director to the knife, the cavity was reached, opened, and a drainage tube inserted. So deep an incision would have been quite impracticable without preliminary puncture, and without the needle to guide the knife.

I had also under my care a woman who presented all the signs of an empyema at the left base. The surgeon was called in and was prepared to operate, but acting on the general rule that the pus should first be found with the needle, I inserted an exploring needle in two or three places and failed to find pus. Under those circumstances the operation was not carried out, and it was as well, for when the patient died, not long after, the pleura was found to be perfectly healthy. The seat of mischief was below the diaphragm, and had its origin in a gastric ulcer, the parts around being all matted together with an accumulation of pus below them, and a general suppurative peritonitis, so that the knife would never by any chance have reached the seat of suppuration.

These cases show the absolute necessity of preliminary puncture before an incision is made. It was a rule laid down, with great emphasis, many years ago by Trousseau, and it should on no account, or under any circumstances, be departed from.

Anæsthetics.—In most cases an anæsthetic will be necessary; certainly if a portion of rib is to be excised.

Chloroform, or one of the chloroform mixtures, is the best anæsthetic to use. Ether, at any rate, should be avoided, on account of the irritation it causes in the large air-tubes. I have seen the most alarming symptoms caused by the accumulation of mucus in the air tubes, as the result of the administration of ether, for the patient, owing to the anæsthesia, was unable to cough it up. Ether is a dangerous anæsthetic in the case of any chest affection.

The amount of chloroform to be given will vary with the operation, but, as a rule, not much is required, for the painful part of the operation is very quickly over.

Local anæsthetics are of very little use, for they do no more than numb the skin and the parts immediately beneath it. Though the freezing of the parts by means of a spray of ether or ethyl-chloride, or the external application of strong solutions of cocain, may suffice for a simple incision of the skin or for a paracentesis, they are of no use for the larger operations. Injections of cocain, or of cocain and morphia, have been advocated, but they are not by any means devoid of risk, and on the whole a little chloroform is by far the simplest and safest anæsthetic to use.

The Preparation of the Side.—The skin of the region where the incision is to be made should be well scrubbed with a nail brush, and washed with soap and water an hour or so before the operation, after which it should be covered with a wet boracic acid compress until the time of operation, when it should be again well swabbed over with some antiseptic solution.

Seat of Incision.—This will be decided in the first place by the needle. Subject to this, where the effusion is localised, the incision will be made as nearly as possible over the centre of the area of dulness.

Where the pus occupies the whole cavity of the pleura, theoretically the incision should be as low down as possible. But it should be remembered that as soon as the pus has been evacuated the side falls in, the lung expands, if it can, and the diaphragm rises; so that, what was the lowest point when the chest was full of fluid, does not remain the lowest point when the fluid has been evacuated. If the incision be made too low down, so soon as the diaphragm rises, the tube does not run straight into the chest, but points upwards and lies for some distance between the diaphragm and ribs.

Again, if the incision be too far back, so that when the patient lies upon the back he is resting on the tube, he will certainly alter his position, so as to take his weight off the tube and the incision, and in this way the tube will no longer be in the most dependent part.

The best place for incision is the posterior axillary region, so that when the patient is lying comfortably on the back the tube will be just free of the bed.

A line drawn transversely through the nipple round the chest will be the simplest guide for the height of the incision, which should be made half an inch to an inch below this line, so that it would pass into the sixth, seventh or eighth intercostal space, according to the position posteriorly which is selected. If a portion of the rib be excised it will generally be one of the ribs named which will be attacked. The sixth or seventh space is the one which is usually found to be opened if these directions be followed, and this is the most convenient spot for drainage and for the general comfort of the patient; and if the needle show that pus can be found here, this will be the place to choose.

Resection of Rib.—The only object of resecting a portion of a rib is to provide such an opening for the insertion of a tube so as to insure free drainage; but if free drainage can be obtained by simple incision through the intercostal spaces, resection of the rib becomes unnecessary, and I suppose the general principle may be laid down that no operation of this kind should be performed unless it be really necessary.

The objections urged against resection are several:

1. It prolongs the operation and renders more of the anæsthetic necessary. This, of course, cannot be denied.

2. Simple incision through the intercostal spaces is a comparatively trivial operation, while resection of a rib is a more serious matter.
3. Pus from an empyema is septic in character, and theoretically it is very undesirable to have septic pus discharging over the recently cut ends of spongy bone. In practice this risk appears to be a very small one. At the same time, I have seen septic infection follow a perfectly antiseptic operation, and even cause the patient's death. Such a result is, however, of such rarity that it need hardly be considered.
4. The chest wall is weakened, but now that the rib is removed by subperiosteal resection, the bone is rapidly restored and the rib replaced in continuity, so that the strength of the side is not ultimately impaired.
5. A free formation of callus may occur, and compress the tube, so that subsequent operations may become necessary on this account. Though very rare, I have seen this occur, and in one case two subsequent operations for this reason became necessary. On the other hand, a formation of bone (which I suppose we ought to call "exostosis") is stated sometimes to occur, even when the rib has not been excised, in consequence of the irritation of the tube. This, however, I have never myself seen.

There are fashions in surgery, and at the present moment the resection of the rib is so much the fashion and the routine practice that few empyemata are opened without it. A difference also exists between the child and the adult; for, while in the adult the intercostal spaces are wider, so that tubes of fair dimensions may be inserted with ease, in most cases, between the ribs, in children the intercostal spaces are narrower, and after excision the ribs fall more readily closely together; resection is therefore regarded as necessary more frequently in children than it is in adults. At the same time, the ribs in children are more yielding and more easily kept separated by slight pressure, so that even if excision be not performed, the tubes are less likely to be pinched.

Even in the case of children, the practice of different authorities varies widely. Thus, in 50 cases Goodhart had the rib excised in 3 only at the time of the operation, *i.e.*, about 1 in 15, while Godlee excised the ribs in 19 cases out of 30, that is to say, in 2 cases out of 3. The results appear to have been equally successful, and it can hardly be supposed that the operation was necessary ten times as often in the one series of cases as in the other; therefore the difference lies greatly in the practice of the individual.

In one series of 22 cases of my own in which the side was opened, a portion of rib was excised in 13 and not excised in 9, and in none of the latter did resection subsequently become necessary.

Of 87 cases at St. Bartholomew's Hospital, the rib was not excised in 10, and in only 1 of these did resection become necessary some weeks later; *i.e.*, in 19 cases out of 109 the rib was not excised, *i.e.*, in about 20 per cent.

Some years ago resection was rarely performed at the time of the initial operation, and subsequent resection was hardly ever required. Recently, following the custom of the day, in a large number of cases the ribs have been excised at once; but I cannot see any advantage in the operation if adequate drainage can be secured without it, nor do I think that the results of recent years are any better than they were before, except so far as stricter antiseptic precautions have been adopted, and this, of course, applies equally to both methods.

I do not, therefore, advocate the routine resection of a portion of the rib. If, when the sides fall in and the ribs come together, drainage cannot be kept complete, resection might be performed then, but most of the cases get well without it, and, I believe, just as quickly.

The hole for drainage, it must be remembered, need not necessarily be round; it may just as well be oval, and as free a vent can be given to the pus by a long incision in the intercostal space, and two pieces of drainage tube laid side by side, as by a round hole which necessitates the removal of a rib.

If a portion of rib is to be resected, the preliminary incision will be made immediately over that part of the rib.

If the incision is to be made in the intercostal spaces, it will be made, of course, between the ribs.

It is never necessary to remove more than a small portion of one rib, about three-quarters of an inch long.

I have seen portions of several ribs removed at the time of the initial operation—an operation which has been described as a "modified Estländer." This is an entire misnomer, for a modified Estländer is no Estländer at all. The object of an Estländer is to provide for the closure of a cavity which will not otherwise close; but no one can tell whether the empyema in a given case will completely heal, or not, until time has been given it. To perform such an operation at first assumes as probable a contingency which is not likely to arise, converts what is comparatively a simple into a considerable operation; being unnecessary, it is bad practice and cannot be too emphatically condemned.

If a portion of rib is to be excised, the resection should be performed subperiosteally, the periosteum scraped off, the bone removed, and with care the whole of this part of the operation may be performed without the pleura being opened.

Washing Out.—When the incision has been made, the pleura should be completely emptied, the patient being, if necessary, turned over on to the affected side in order to allow all the pus to run out. If the pus be fœtid or thick and curdy, the pleura should be washed out with a warm solution of boracic acid. Care must be taken in doing this that the fluid does not accumulate in the pleura, but that there is a free vent, so that it may flow out as fast as it flows in.

Washing out the pleura is often objected to as being a risky or even dangerous operation. This is not the case. I have washed the pleura out myself, I should think many hundred times, and have never seen an accident of any kind occur. When I first became interested in chest affections, washing out the pleura was the routine and daily practice, and such an experience demonstrates, at any rate, the safety of the procedure. With free drainage it is rarely necessary, however, to wash the side out more than once, viz., at the time of the operation. It will only require repetition now and then if the pus be fœtid, and not always then. At the same time, there can be no objection on the ground of risk to washing out whenever necessary. It is quite true that disastrous cases have been recorded, and have been attributed actually to the washing out. Most of these cases have been instances of sudden death; but it must be remembered that death may take place suddenly in pleural effusions which have not been washed out, and have not been even incised. The subject has been already discussed, and there are probably other causes, entirely independent of the washing out, which explain it.

The only precautions necessary in washing out the pleura are, that the fluid should be warm, and the vent free, so that the fluid may flow out readily. It is obvious that if the fluid be allowed to accumulate in the pleura, pressure may be exercised upon the lung, vessels, and heart, and in this way harm may be done; but with these precautions and ordinary care, washing out the pleura is perfectly free from risk.

If the pleura contains curdy or flaky pus, blood clots, or fœtid fluid, washing out is the right thing to do, and will greatly expedite recovery.

Counter Opening.—A counter opening is rarely made now, and is, indeed, seldom necessary. The object of it was to provide free drainage, but this can be generally obtained by a single opening made in the right place. Sometimes it happens, when the cavity has almost completely closed, that there is a small cavity left in another part, behind possibly, which does not freely drain through the opening, and it may be necessary to make a fresh incision there and put in a tube, allowing the one in front to close; but this, again, is very rarely required, and can usually be avoided by proper management of the tube, as will be referred to later.

Sometimes, when the empyema has already spontaneously discharged in an inconvenient position, *e.g.*, in the upper and front part of the chest, it may be necessary to somewhat enlarge this upper incision, at the same time that another is made in the ordinary position, and it may be convenient to pass a drainage tube through the chest from the one to the other. In this way, no doubt, cure is expedited, the upper opening being, after a time, allowed to close, as soon as drainage through the lower one is complete. In such cases it is as well to use the upper opening for the introduction of a probe, so that it may be ascertained that there is a really free communication with the lower opening where the new incision is to be made.

It may happen that there are two or more separate collections of pus in the pleura, so that one incision will not drain both. Then more than one incision will become necessary ; but these cases are exceptional.

The Tube.—If a portion of rib has been excised, a tube half an inch in diameter may be inserted ; if not, two tubes of smaller diameter placed side by side will be sufficient.

The tube should in any case be of such a length as to reach nearly to the bottom of the cavity. Where the whole pleura has contained pus, it will possibly require to be 4 or 5 inches in length at first.

In any case the greatest care should be taken to see that the tube is securely fixed, so as to avoid any risk of its slipping into the side. For this purpose it is well to pass a strong thread right through the tube, and fix it to the side by strapping. If by any chance the tube should slip into the side, it will be a source of considerable danger, for if not removed it will almost certainly set up some grave septic changes, while to find and extract a tube from the pleura, through a small incision, is an operation of the greatest difficulty and is frequently unsuccessful.

I have only once had such an accident happen in my own experience, and then fortunately the tube was recovered, but there are many cases on record and several of them proved fatal.

I saw a child aged 9, who had been operated on for empyema successfully nine months previously. The wound healed in a month, but after six months the discharge reappeared, and three months later she came under my care. On dilating the orifice of the fistula, a piece of drainage tube was found lying close beneath, which must have been in the side for nine to ten months. It was removed, and the child quickly got quite well.

In the same way a good deal of care must be taken not to apply loose lint, gauze, wool, or such easily detachable substances immediately over the wound for they are likely to get sucked in with inspiration, and may then retard recovery for a considerable period of time. This danger is often not mentioned, but it is a real one, and should be present to the mind when the dressings are made.

The Dressing.—There is little to be said on this point, for any of the ordinary surgical dressings may be used, the only precaution necessary being that just mentioned, viz., that there should be no fluffy stuff, or anything that could be easily sucked into the pleura, lying immediately over the wound.

The dressing should be changed as soon as the discharge has come through. It is difficult to say when this will be, but as a rule the dressings will have to be changed pretty soon in ordinary cases, perhaps two or three times in the first twenty-four hours. The discharge is often very profuse for the first two or three days after excision, but its bulk is chiefly made up of serum, which is freely exuded from

the pleura. The admission of air to the pleura is no doubt a violent irritant to it at first, but as soon as the irritation subsides, the discharge becomes greatly reduced in quantity, and may in a few days be reduced to a very small quantity, so that the dressing may even be left on for several days without removal.

It is well, however, to look at the incision from time to time, for if the tube be not properly fixed it is likely to slip out or be forced out by coughing, and we may then find that the wound has closed, and another one may have to be made. Indeed, I think it well, even if the dressings be not changed, for the side to be looked at every day to see that the tube is *in situ*.

Rashes after incision occasionally occur, as they may after paracentesis. They are generally of the nature of erythema or urticaria, and may be compared with the similar rashes which are sometimes seen after tapping the abdomen or a hydatid of the liver. They are of no importance, and do not affect the course of the case in any way.

Thus, in a boy of 3, two days after the side was opened a general erythema developed, somewhat like the rashes described as surgical scarlet fever, or, more correctly, septic erythema. This disappeared without any trouble resulting. In another instance, a woman of 30, the operation was followed by troublesome urticaria, which recurred three times, and was related, I think, in some way with the washing out which was practised, for the patient had carboluria also.

The Mode of Cure.—The cure of an empyema is only complete when the lung and the chest walls have come into contact again.

Usually this is attended by adhesion of the two layers of the pleura together, so that the cavity of the pleura is completely and permanently obliterated; yet adhesion need not necessarily take place, and probably in some instances does not, as is shown by cases of recurrent empyema, in which a second empyema occurs some time afterwards upon the same side.

As soon as the pus has been removed from the pleura, the cavity becomes greatly reduced in size. This is brought about, first, by the falling in of the ribs; secondly, by the rising of the diaphragm; thirdly, by the return, to some extent, of the heart and mediastinum; and lastly, by the re-expansion of the collapsed lung.

1. The diaphragm rises immediately the side is opened. This is due to its being forced up by the abdominal contents under the contraction of the abdominal muscles.

2. The chest walls fall in. When the collection has been a large one, and the side has been much distended, incision relieves this distension, and allows the ribs to fall back to an intermediate position at once. But in some cases, even where the effusion is large, the side is not distended, but somewhat flattened and contracted, and this flattening always takes place in the course of time after an incision has been made.

The extreme contraction which follows a long-standing empyema or a chronic pleurisy is, no doubt, to be explained by the contraction of the fibrous tissue which has formed between the two layers of the pleura and the root of the lung; but considerable contraction of the side is often seen when the lung and the chest walls are not in contact, so that some other explanation must be sought for. The contraction is most marked in the lower part of the chest, and this is to be explained, no doubt, by the pull of the abdominal muscles upon the lower ribs, which tends to flatten them; and this downward pull, no doubt, tends to flatten the whole side, and the upper ribs as well, to some extent. But another factor, which I think comes into play, is the palsy, from disuse, probably, of the intercostal and other muscles, which, when healthy, tend to keep the ribs raised. This part of the flattening, which is due to loss of muscular power, could easily be removed when the muscles come again into action, and thus we have an explanation of the rapid disappearance of a good deal of the flattening when the incision has closed, as well as the appearance of flattening before the fluid has been removed from the pleura, which has been already referred to.

3. The re-expansion of the lung. We are usually told that when the side is opened, the lung contracts by virtue of its own elasticity, which is sufficient in a short time to produce even complete collapse of the whole organ. If this be the simple fact, it is difficult to understand

how the lung ever expands again. The explanation of its expansion usually given is that adhesions form between the lung and the pleura, and these, by their contraction, slowly pull the lung out again ; or that the cavity granulates up from the bottom and closes in this way, and that subsequent contraction of these granulations causes the lung to expand. When one really considers these explanations, they seem to be impossible, nor is there much evidence to support them ; for, in the first place, the surfaces are not covered with the luxuriant granulations that this theory would suppose, and connective tissue, when it forms, is quite as likely to bind the lung down as to form between the two layers of the pleura and cause it to expand. Indeed, it requires but little clinical observation to see that this explanation is not correct.

Within a very short time of the incision the collapsed lung begins to expand and to resume its respiratory functions again, and sometimes within a day or two it is found not far from the external opening in the thorax, while there are some cases of empyema in which, after incision, the tube has been removed and recovery has taken place within a comparatively few days—two or three weeks, or even less. Of course, where the tube has been removed and the external incision closed, leaving a certain amount of air within the thorax, it is possible that this air may be absorbed, and the forces of absorption may come in to help the lung to expand. But the lung is very often found to be expanded, and to be lying close beneath the walls of the thorax, even when the incision still remains patent.

No doubt forced expiration and coughing assist to expand the lung, but these forces are intermittent in action, while the expansion of the lung is persistent.

The only conclusion that remains appears to be that a collapsed and compressed lung rapidly loses its elasticity. This conclusion seems, on its own merits, to be improbable.

Any way the fact remains that the lungs do expand with very great readiness, and in a way that is not in accordance with the current theory of their elasticity.

In children the cure of empyema is most rapid and the most complete, and at the same time it often happens that the deformity is greatest. These two facts are to be explained, no doubt, by the same conditions, viz., the softness of the sides of the thorax. Even after very considerable deformity in children the chest may, in time, regain perfect symmetry.

In these cases the recovery may be complete, and there may be no displacement of organs, and indeed nothing except the scar of the old incision to show what had been the matter.

In other cases, though the external deformity be rectified so that there is no obvious want of symmetry externally, still the deformity has been rectified by displacement internally of the organs, so that the heart is found beating a long way out on the affected side, the mediastinum is displaced, and the opposite lung has undergone considerable complementary hypertrophy.

In the adult the chances of complete recovery are not so good ; that is to say, more or less deformity (flattening) is almost certain to be left. Besides this, the chances of complete closure of the cavity are not so great, for the lung in the adult is so frequently diseased.

The After-management of the Case.—The frequency of dressing will be determined by the amount of discharge. That has already been referred to. The rest of the management consists in securing perfect drainage through the tube, and in shortening the tube and getting rid of it as quickly as can safely be done.

As the cavity diminishes in size, the tube may be shortened in diameter and diminished in calibre. It is not desirable to be in too great a hurry to get rid of the tube. If the tube be removed too soon, the pus will re-accumulate ; and if the site of the opening has closed, so that the pus cannot make its own exit, a fresh incision will have to be made and the tube reinserted. It is a great mistake to be in too great a hurry, for to leave out the tube is risky, and will very often necessitate a fresh operation. I have seen no less than three different operations necessary in the same case because the tube was removed too soon. At the same time patients can go about their business, in the later stages, with a small tube in their side, without any discomfort or disadvantage.

If the tube cannot be removed within the first two or three weeks of the operation, the rule that should be laid down is that it shall not be left out until it cannot be kept in; that is to say, it should be forced out by the expansion of the lung, or by the granulations filling up the track of the tube from the bottom.

Of 134 cases which were operated on and recovered, in 20 only was the tube out, and the wound healed, within six weeks from the time of operation; of these the great majority (viz., 16) were children, 5 being infants under 2 years of age, 9 under 10 years of age, and 2 aged 13 and 14 years respectively. The remaining 4 were adults of the ages of 19, 30, 50, and 60 years.

Of the rest, *i.e.*, 85 per cent., the majority had the tube in the side for varying periods of time beyond the six weeks. Though the tube could not be dispensed with entirely for some weeks or even months longer, the side was practically well, the lung had expanded completely, and all that was left was a small sinus, or possibly a small cavity which did not completely close.

In a few instances the tube could not be got rid of for a year or two. Even in these cases recovery was ultimately complete.

There are sometimes special reasons in the track itself why it will not heal; *e.g.*, I think they not infrequently become tubercular. In such cases, whether tubercular or not, the sinus should be treated with some active disinfectant, for instance, creasote dissolved in oil.

I have seen one case in which the discharging sinus had existed for eighteen years. Such a result must be referred to neglect or improper management of the empyema in its early stages, and is hardly ever likely to be met with at the present time.

The tube may sometimes be removed very soon after operation. The following are the cases of most rapid cure which I have met with:—

In 12 days in two boys of $6\frac{1}{2}$ and 9 years of age.
,, 13 ,, in a boy of 2.
,, 14 ,, in a boy of 8 and a girl of 8.
,, 17 ,, in a boy of $2\frac{3}{4}$.
,, 18 ,, in a man of 50.
,, 24 ,, in a boy of $2\frac{1}{2}$.
,, 28 ,, in boys of 1, 5, 8, and 3 respectively, and in girls of $1\frac{1}{2}$ and 14.
,, 28 ,, in a man of 30, and in two women of 19 and 60 years respectively.

How long the tube will have to remain in, it is impossible to say in any given case. It may be for a considerable time—weeks or months, and sometimes even years; the age of the patient, duration of the empyema before operation, and the nature of the case have all to be taken into calculation; but, as a rule, the prospects of rapid recovery are best in children.

The young lady to whose case I have referred, where three operations were necessary to reinsert the tube, was not able to dispense with it for three years after the original incision. The patient then, however, became completely well, and had no trouble or difficulty afterwards.

The after management of the tube is not by any means so simple as is often thought, for it does not follow, because the tube is in the chest, that it is in the cavity and draining the cavity properly; for the lung, not infrequently, expands irregularly, so that in the course of time the tube comes to lie in a fistulous track of its own, and may possibly be completely surrounded by the re-expanded lung, and be no longer in the cavity of the pleura at all. The tube may actually cross the cavity, and by doing so close the aperture into it; the patient will then get the signs of pent-up pus, the most obvious of which is generally a rise of temperature; and every time the tube is removed a certain amount of pus will be evacuated, showing that drainage has not been complete. These cases should be carefully examined with a probe, bent slightly at the end. With a little manipulation it is not difficult to find a way into the cavity, and to discover that the tube does not lie in it. A little manipulation will then enable the tube to be

passed along the director in the proper direction, so that the cavity can be completely drained. Indeed, it should be the rule, in a chronic case of empyema, to examine the cavity that is left with a probe from time to time, so as to know exactly what the conditions are which have to be dealt with.

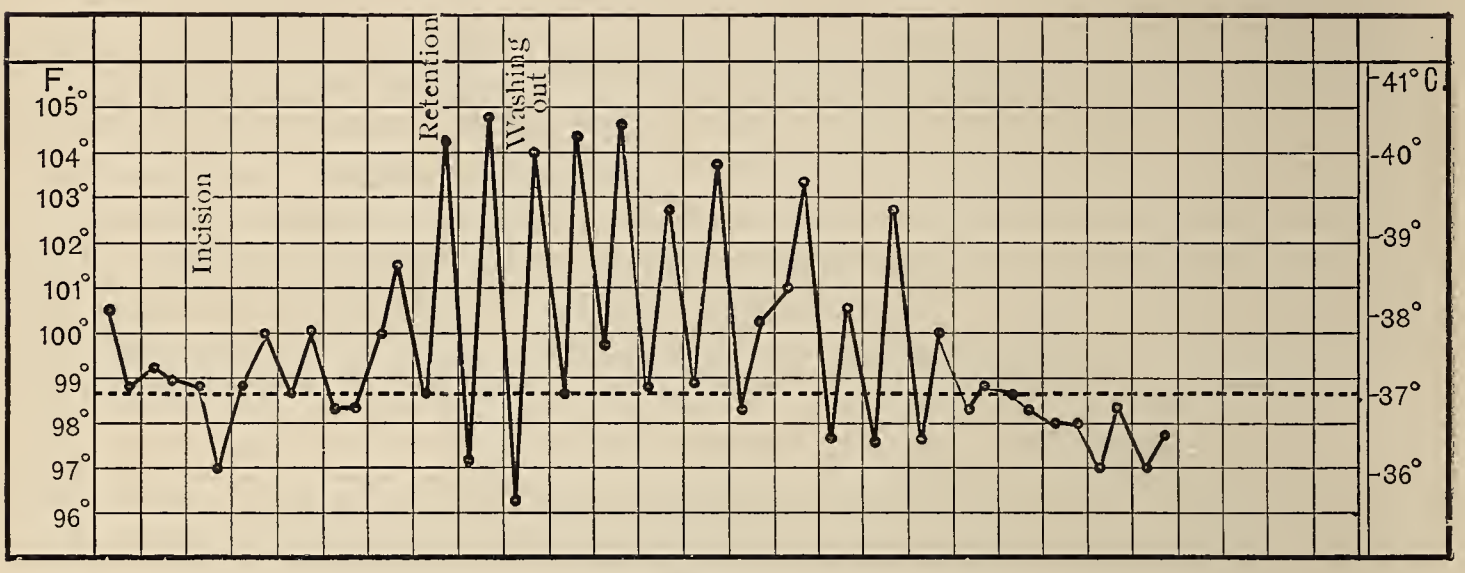


Fig. 152.

Empyema, left side. Opened. The chart shows—1. The fall of temperature after incision. 2. The difficulty, even with a free incision, of maintaining free drainage, as shown by the hectic. 3. The fall of temperature, when drainage was complete.

Cases in which there were Difficulties with the Tube.—A lad of 12 years of age had the left side freely opened and a piece of rib excised, with the removal of 6 ounces of curdy pus. A few days later a copious discharge of pus occurred suddenly, showing, no doubt, that the original incision did not reach the main collection of matter. The cavity was washed out, but still the drainage was imperfect, and the temperature continued raised. On examining the pleura with a long probe, the cavity was found to extend a long way towards the back, and a tube 5 inches long was inserted. This led to a free evacuation of pus. The temperature, which had been very irregular, became normal, and from this time recovery was rapid. In about six weeks' time the cavity had completely closed, except for the short sinus in which the tube lay.

A girl of 5 years of age, with left-sided empyema, had been operated on, pieces of several ribs excised in the posterior-axillary region, and a very large hole made into which a large tube was placed ; but a few days later it was evident that, in spite of the size of the tube and of the wound, the cavity was not draining properly. Examination with the probe showed the tube to lie in a fistulous track, and to cross rather than enter the cavity. This was then rectified, and recovery was rapid.

A boy of 3 years of age, with right-sided empyema, was operated on in the usual way, and a portion of the sixth rib excised. A few ounces of curdy pus were removed. Examination with the probe, a week or two later, showed that a large shallow cavity existed, half an inch deep but 6 inches long and 4 inches wide, lying beneath the ribs. Six months later the external wound was enlarged, and the cavity proving to be as extensive as before, a counter opening was made behind at the lowest part, and three-quarters of an inch of the corresponding rib removed. Much pus escaped at the time of operation, and rapid contraction of the side followed. In a couple of months nothing was left but the track in which the tube lay. Eighteen months later an abscess formed in front of the old sinus in which the tube lay. This was incised, and the posterior wound closed ; but soon after it broke open again, and the anterior opening closed. The child still had, three years from the first operation, the tube in the side, though it lay in a small sinus only. Ultimately, in the course of some months, the sinus healed completely.

It is often objected that, if a tube be left in for a long time, it may cause troublesome ulceration, caries of the rib, hæmorrhage, and other complications. I have seen some of these events occur, but not of recent years, and with our modern methods I do not think these complications are at all likely to be common. With ordinary common-sense management the tubes may remain in, as I have stated, for months, or even years, without any disadvantage.

The results of operation are eminently satisfactory; and there has been remarkable improvement of recent years. This is due, no doubt, partly to improved technique and stricter antiseptic precautions, but chiefly to the fact that the cases are operated on much earlier than they used to be.

Goodhart's[1] statistics for 77 cases give a mortality of 44 per cent.; but the general mortality for 254 cases at St. Bartholomew's Hospital for the twelve years (1883–94) is only 21·5 per cent., and no doubt the difference in the main may be rightly attributed to operation; and I have little doubt that even these results are being improved upon every year.

To determine the actual mortality and risk of the operation, I have carefully analysed 156 cases, of which about 50 have been under my own care, and the rest are taken from the records of St. Bartholomew's Hospital for the five years 1890–94 inclusive.

Out of these 156 cases 27 died, which gives a percentage mortality of 17·3; 9 of these were infants (*i.e.*, 2 years of age or under), and as the mortality in infants is unusually high, these may be deducted, and the mortality for the rest of life is reduced to 11·5 per cent.

Of the 156 cases 10 were not operated on at all, or at the most had paracentesis only performed.

Of these 10, 4 were infants, 2 of whom were practically moribund on admission, and 2 others died of meningitis and pyæmia respectively.

Of the remaining 6, chiefly adults, 4 died of pericarditis, 1 with pus in the anterior mediastinum, and 1 with cancer. Those 10 cases were, therefore, unsuitable for operation.

Of the remainder, 146 cases, 17 died, yielding a mortality of 11·6 per cent.

5 were infants, and if these be deducted, the mortality for other ages is reduced to 8·5 per cent.

Of the 12 cases left, 7 died of causes not in any way connected with the operation, 4 died of phthisis at various periods after operation, 1 of meningitis, 1 of morbus cordis, and 1 after a second operation involving the removal of portions of several ribs, the patient being also phthisical.

Thus there are left 139 cases with 5 deaths, yielding a mortality of 3·6 per cent. This, I believe, represents the actual mortality after operation in uncomplicated cases.

Modern methods have, therefore, produced the same improvement in the operation-statistics for empyema as they have for other operations. I think the mortality will be still further reduced when the importance of the careful after-management of these cases is more clearly recognised than it generally is at present.

If the cavity does not close, what else can be done?—If the cavity does not close, the patient is left with a fistula, from which more or less discharge takes place. This condition is regarded by many writers as a dangerous one; first, because there is a constant risk that the fistula will become obstructed, and pus accumulate in the cavity; secondly, because septic changes may be set up and the patient become seriously ill in consequence; and lastly, because if the discharge last a considerable time, amyloid disease may develop.

The first two risks are successfully obviated by providing free drainage, and as I have already stated, patients may wear a tube for years without any ill results, provided that a free vent be provided for the discharge.

It may, however, be found, in the course of time, after the cavity has contracted to a great extent, that the opening is no more in the most dependent position, and drainage, therefore, is not complete. If that be so, the cavity should be examined with a probe, an incision made in its lowest part, and a fresh drainage tube inserted. This, if it does not lead in the end to cure, will at any rate provide complete drainage.

It must be borne in mind that in cases of long-standing fistula the difficulty often lies, not in the pleura, but in the fistulous track itself. The pleural cavity has long ago healed, and examination with the probe shows that there is no longer any communication with the pleura; but whenever the tube is removed from the fistula, the mouth closes, pus accumulates below, and ultimately discharges itself by the old opening. This is just what is seen in any other sinuous

[1] *Guy's Hosp. Rep.*, 1877.

fistula, but if treated on general surgical lines, and made to granulate up from the bottom, they can generally be made to heal.

The risk of amyloid disease is, I think, very greatly exaggerated. It is only likely to occur where a very considerable discharge takes place day by day, and not always even then. As a matter of fact, even in chronic cases of empyema, the amount of pus discharged daily is very often quite small, not more than a few drachms, and this is no more likely to produce general amyloid disease when it takes place from the pleura than it is when it occurs from any other part of the body, as, for example, from a tubercular gland in the neck or elsewhere. Amyloid disease presupposes an excessive suppuration.

Where a fistula has existed for a long time, *i.e.*, years, there is some little risk in interfering with it, for the patients are not in a good condition to bear interference of even a simple kind, and they sometimes come to grief when there did not appear to be any special reason why they should.

A patient came under my care who had had a discharging empyema for fourteen years. The fistula was a tortuous one, through which I could not get a probe to pass, and I had an incision made and a portion of rib removed for the purpose of drainage. The patient had a rise of temperature following the operation, and died in two days, having been apparently in fair health until the operation. The operation, no doubt, was the cause of his death, and experience has led me to be cautious how I interfere with cases of this kind in any radical way.

On the other hand, recovery is sometimes very rapid and complete after operation. Thus I had a young soldier under my care who had acquired an empyema which burst spontaneously through the second intercostal space in front as well as through the lung. He was discharged from the army as incurable. Six months later he came under my hands. I opened the fistula existing, and made a counter opening as far down and to the back as I conveniently could. Before the operation he had been spitting a pint of pus daily through the lung, and almost as much had been discharged by the fistula. In a week after the operation the expectoration ceased, and in five weeks the tube was out and the side healed. The lung re-expanded completely, though it had been compressed for nine months or more.

When the cavity of the pleura will not close, the best method of dealing with it must be determined by consideration of the condition of the side in that part. This necessary knowledge can only be ascertained by careful examination with a probe. The cavity left in these long-standing cases is, as I have stated, frequently a shallow one, possibly not more than half an inch or so in depth, but extending a considerable distance beneath the ribs ; it is often irregular and has diverticula, and is frequently found to be somewhat deeper at the back than in the side and towards the front. A thorough knowledge of the conditions to be dealt with is necessary before the right method of operating can be determined. It is, of course, obvious that it would be useless to remove portions of rib in the axilla if the cavity that we had to close were at the back.

If the cavity will not close, there are but two courses—either to leave it alone for nature to cure in its own way in the course of time, or to perform an extensive operation, viz., that of Estländer.

Estländer's Operation—Thoracoplasty.

Estländer's operation consists in the removal of a wedge-shaped piece from the wall of the thorax, and involves the excision of portions of several ribs. The piece removed often measures many square inches in area, being an inch or so wide at the top, and, it may be, as much as eight or nine inches long at the bottom, the ribs being removed to a corresponding extent. It is a plastic operation ; its rationale rests on the assumption that it is the rigidity and resistance of the ribs which prevent the side from falling in sufficiently to close the cavity, and its object is the removal of this resistance.

The indications for the operation are the presence of a cavity of some size and good grounds for believing that it cannot close without an operation of this kind.

Some time, many months at least, must be allowed to elapse before it will be evident that the cavity will not spontaneously close, and it is astonishing what nature will do with time and patience, even in apparently very bad cases. There is, therefore, no need for hurry in deciding. Again, something will turn upon the condition of the lung, *i.e.*, whether the lung be capable of re-expansion, and to what extent. Where the empyema has been allowed to exist for some time before operation, the lung may become bound down in its collapsed condition by dense fibrous adhesions, and so be incapable of re-expansion ; but this can by no means be assumed, for in many cases the lung expands quite readily when the fluid is removed, though *a priori* the chances would seem against it.

The objections to the operation are obvious.

1. It is an extensive and very serious operation.
2. Under the most favourable circumstances, it will greatly diminish the strength of the thorax.
3. Even when successful, it will be attended with very great deformity.

It will have, therefore, to be very seriously considered in every case, first, whether so extensive an operation is desirable, and secondly, whether the result, if it prove successful, will justify the operation.

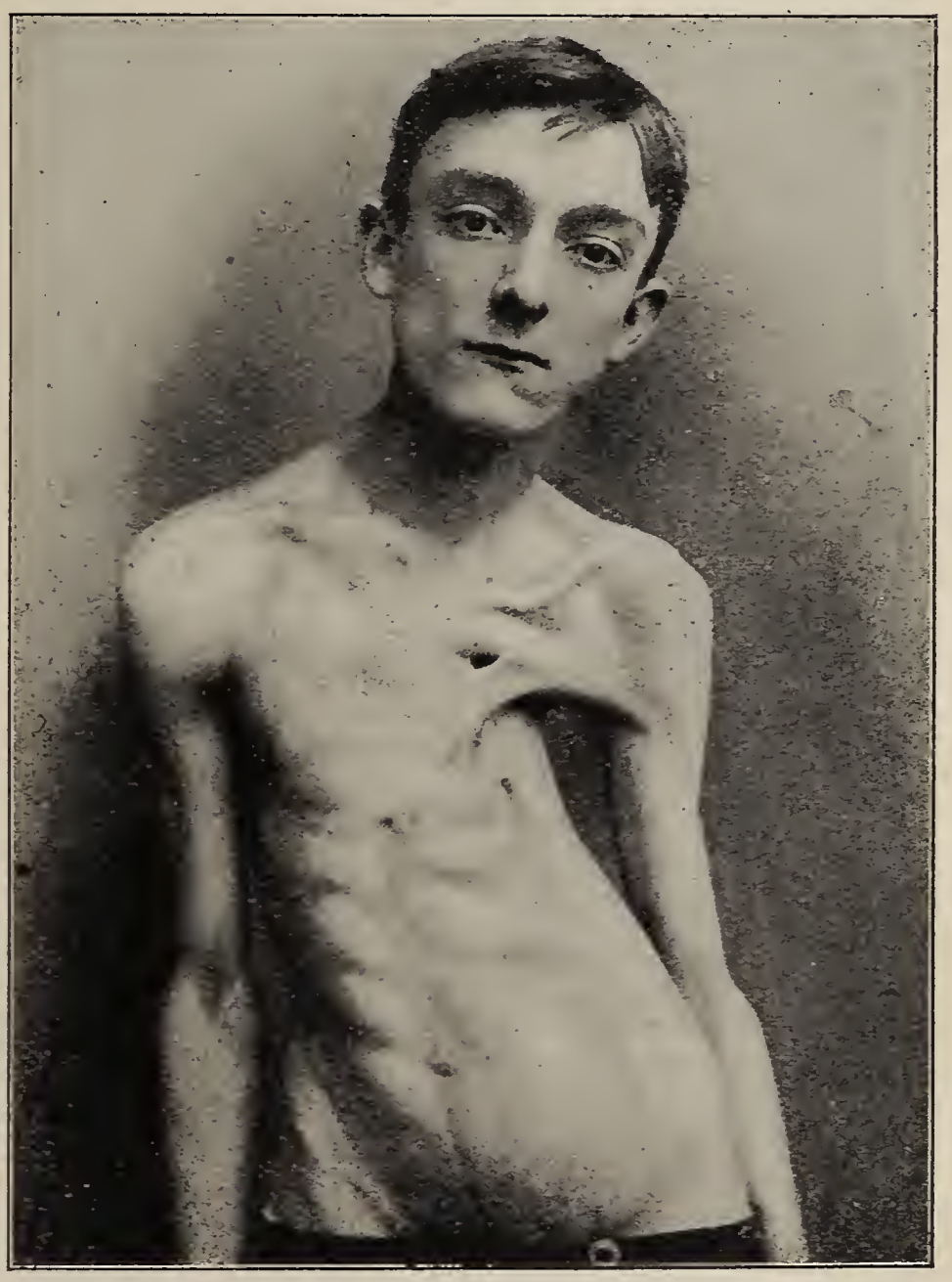


Fig. 153.

The operation is a serious one. Many cases die as a result of the operation. Few of the fatal cases are recorded, so that no opinion can be formed of the full risk of the operation from the statistics of published cases. The operation should not be undertaken at all, except in patients who are in such a condition as to render success probable in respect of their general health at any rate ; yet when the general health is well maintained the operation is usually not thought of, and when the health is broken down the operation is clearly unsuitable; especially is this the case if the patient be phthisical, or if the lung be otherwise diseased.

The deformity left behind is considerable, and is well shown in the accompanying illustration, which is a photograph taken of a patient operated on by Mr. Pearce Gould seven years previously.

This may be regarded as a very successful case, for at the time of the operation there was a profuse discharge, the liver was enlarged, the general health much broken down, and the patient seemed to be in a condition of advanced amyloid disease. Drainage had been fully tried and failed. After the operation, the patient's general health was quickly restored, and a complete contraction of the cavity took place, but with the result shown in the photograph.

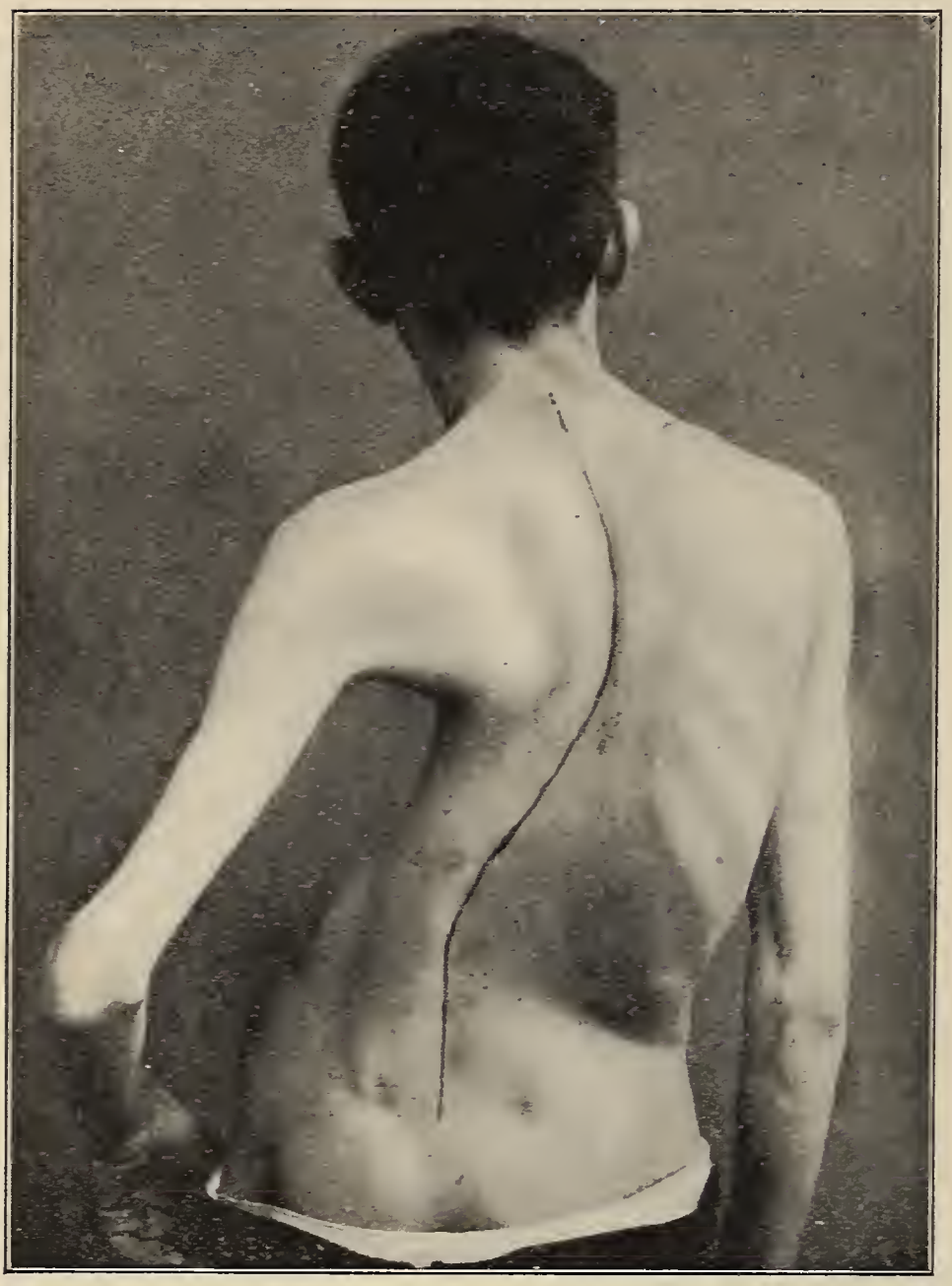


Fig. 154.

The Operation.—If the operation be decided on, before anything is really done the cavity must be carefully explored, so that its extent and whereabouts may be fully realised.

This may be done with a probe, before the operation, or by a finger inserted in the thorax at the time of the operation.

Anyhow, the position of the incision and the extent of the excision of ribs will be determined by the extent and position of the cavity; for to ensure success it is necessary that the bony and resistant parts forming the outer wall of the cavity should be completely removed. This may necessitate a very large and formidable operation, but to be successful, it must be complete, or else it had better not be undertaken at all.

What is sometimes called a "modified Estländer" is no Estländer at all; indeed, it runs exactly counter to the very principle of the operation, and is really nothing more than an exaggerated and unnecessary drainage operation.

The seat and extent of the operation having been determined, the flaps should be reflected and the ribs removed by sub-periosteal resection. This disturbs the position of parts as little as possible, and avoids a great deal of hæmorrhage, for with care this can be performed without division of the intercostal arteries.

The number of cases recorded is but small. Up to the year 1885 Ceccherelli[1] was able to collect only 61, the results of which he tabulates as follows :—

Cured,	15	Died from operation,	8
Much improved,	13	Result unknown,	20
Made worse,	5		

In some of these cases the operation was a comparatively small one, and many of the cures are found in this group. On the other hand, 50 per cent. were not improved, or were made worse, and of these 12½ per cent. were actually killed by the operation. To these must be added an unknown number in which the operation failed, and no record has been published.

Cases suitable for such an operation will always be rare, though as with all new operations the numbers at first seem to be considerable, owing to the accumulation of such cases over a period of many years. We may confidently anticipate that with the earlier treatment and better after-management of empyema, the cases will become fewer and fewer in which a plastic operation is necessary.

Under any circumstances, an operation of such magnitude as this should only be undertaken by those well accustomed to antiseptic surgery and with considerable experience in operations upon the chest. It is a serious operation, for the shock which follows it is considerable, and the risks, therefore, not to be despised; and it is performed under great disadvantage where the general health is undermined, and the patient hardly in a condition to stand so severe a strain.

Among the 156 cases of empyema collated, there were 3 in which a thoracoplastic operation was performed. One of these was performed within fourteen days of the original incision, and can hardly be rightly put into this category. The other two were performed about six months after the opening of the side. In the one, a lad of 14, portions of five ribs were removed, and rapid recovery followed, nothing but a track in which the tube lay being left in the course of four or five weeks.

In the second case, a man of 25, six months after the first incision, about an inch of each of the fourth, fifth, sixth, and seventh ribs were removed in the post-axillary region. This led to rapid contraction of the side, but a fistulous track remained nine months later.

The Treatment of Empyema by Paracentesis.

The admirable results of the treatment of empyema by free incision have caused paracentesis to appear antiquated and out of date; but if it be true, as has been stated, that most cases of pneumococcus empyema, and many of streptococcus empyema, may be cured by simple paracentesis, it is possible that paracentesis may come into fashion again.

It is the general rule in surgery that when pus is present in the body it should be given free vent as soon as possible, and no doubt the same general rule applies to the pleura. Yet it must be acknowledged that the pleura is a special cavity, and requires peculiar consideration, and I suppose it will be conceded that to open the pleura, at any rate in an adult, is no trifling operation, and may involve fairly serious consequences.

Paracentesis certainly suffices in some cases to effect cure, and there are many cases on record in which even a single tapping has been sufficient; many more in which, after two or three tappings, recovery has been complete; and a few in which systematic tapping over a series of weeks or months has resulted in final cure.

The most remarkable case of the latter kind is recorded by Lilly.[2] In this patient, a man of the age of 18, paracentesis was performed in all thirty-two times, almost daily for the two last months. In the end this patient made complete recovery.

The case is remarkable in another respect, on account of the large amount of pus that was removed at many of the paracenteses.

[1] *Virch. Jahrb.*, 1885, ii. 147.

[2] *Glasgow Med. Journ.*, 1872, 44.

Thus, on the first occasion 130 ozs. were removed ; on the second, 105 ; on the fourth, 155 ; and after this, when the paracentesis was more frequent, the quantities became less and less, until ultimately cure was complete. I do not suppose that cases of this last kind are of any great interest at the present time, except as showing what is possible, for if improvement does not take place after two or three paracenteses at the most, the side would, nowadays, be treated by free incision, and, I think, quite properly.

Most of the cases of successful treatment by paracentesis have occurred in children, and if in the adult, usually in cases of localised empyemata. Yet in the adult, in cases even of considerable effusions, paracentesis is sometimes successful.

1. Thus I know of the case of an adult of about 30 years of age, in whom there was a very bad family history of phthisis.

   It was decided not to lay the side open, but to employ paracentesis. On the first occasion about 2 pints of pus were removed ; two or three weeks later a second paracentesis was made, with the removal of about 8 ounces of pus. On a third occasion, only about 1 ounce was obtained, and in a few weeks the patient was completely well. I understand that now it is difficult to tell by physical examination which was the side affected, or that there has been any disease of the kind at all. This occurred many years ago, and the patient still remains in good health.
2. In another instance, a case of my own, a woman of 30, who had been recently confined, and who dated her illness from the confinement, was found to have a large pleuritic effusion. She was desperately ill, and it was thought would die. She was immediately tapped, with great relief to her symptoms. She improved much after this, and in the course of three weeks or so, when the fluid seemed to be stationary, it was decided to have the chest opened ; but when the day for operation arrived, it was thought wiser to postpone it, and instead of incision the patient was tapped again. This was followed by rapid recovery. In this case the patient when first seen was so run down and so feeble that it was thought expedient to postpone the question of operation, and to perform paracentesis as a palliative measure. I have little doubt that, if the side had been opened then, the operation would have proved fatal ; and even at the time the operation was decided upon, her general health was still so bad that I was glad to have an excuse for postponing incision. From the character of the case I have little doubt now that it was a streptococcus empyema.

In localised or encysted empyema there is not the same objection to free incision, and such cases can be treated as well by free incision as by paracentesis ; indeed, it is free incision that I prefer. However, in localised empyemata it is not always easy to localise the pus, and it always has to be discovered by means of the needle first, and then we sometimes find that removal of pus by the aspirator suffices to effect cure.

3. Thus a healthy man of 35 came under treatment for what, it was thought, was an empyema. There was a considerable amount of dulness on the lower half of the right side. On the twelfth day of his illness a needle was inserted, but no pus found. Many punctures were subsequently made, with the same want of success, and it was not until an anæsthetic was given, and several punctures made on the same occasion, that pus was ultimately found. Nine ozs. of thick curdy pus were then removed with the aspirator, and from that day the patient made a rapid and uninterrupted recovery.
4. Another case was a child of $3\frac{1}{2}$ years of age. The empyema apparently followed an attack of measles, and had existed for about three weeks before she was admitted into the hospital. The empyema seemed to be a fairly large one, for there was dulness from the base up to the level of the third rib. Fourteen days later a needle was inserted, the aspirator was used, and 2 ozs. of sweet, very viscid pus removed. The needle appeared to be in a very large cavity, but no more pus could be obtained, and on subsequently examining the instrument, the tube was found to be choked, so that there was but a very small portion of the effusion removed ; still, four days later, it was evident that the fluid was diminishing, and in a fortnight the greater part of the effusion had completely disappeared. Three weeks later, that is to say, in about six weeks in all after admission to the hospital, air entered quite down to the right base, and the patient seemed to have made complete recovery, though there was a little impairment of percussion still at the lowest part of the chest behind. The child was sent away to the seaside, and when she returned seemed to be quite well. This case is very interesting, for it shows that even a large amount of pus may be completely absorbed from the pleura.

It may happen that after paracentesis the fluid may change its character, that which re-accumulates being less purulent than that previously removed. I have already referred to one case of this kind when speaking of paracentesis for serous effusion. The following is another instance of it.

5. A man of 37 was tapped, and 80 ounces of pus were removed from the left pleura. Twelve days later the needle showed the presence of sero-purulent fluid only, and this was rapidly absorbed, with complete recovery.

The following additional cases are also taken from my note-books :—

6. A boy of 5, with left-sided empyema, following broncho-pneumonia after whooping-cough and measles, was tapped twice at intervals of a few days, with the removal of 6 and 5 ounces of pus respectively. The temperature fell after paracentesis, and the child got perfectly well, with some contraction of the side.
7. A girl of 13, with right-sided empyema, came under observation after one month's illness. She was extremely ill, with a hectic temperature reaching 103° every evening. Paracentesis was performed three times ; 15 ounces were removed on the first tapping, 6 ounces on the second two days later, and 1 ounce only on the third a fortnight later. Recovery was rapid, and the child gained 10 lbs. in weight in fourteen days. Great contraction of the side took place, but two months later all trace of deformity was gone.
8. A man of 33, who had been ill a month, presented the signs of empyema at the right base. The temperature was not raised when seen. Paracentesis in the mid-axilla removed several ounces of thick pus, after which recovery took place.

The cases which follow are for the most part taken from the records of St. Bartholomew's Hospital :—

9. A lad of 16 was attacked with pneumonia. The temperature fell after the crisis, but rose again gradually. Signs of fluid appeared on the left side, and the pleura was tapped twice, with the removal of 8 and 15 ounces of pus respectively. The temperature became normal, and the physical signs almost completely disappeared.
10. A boy of 8 had been ill for two months, and presented the signs of fluid on the lower part of the right side. On paracentesis only a little pus was obtaincd, the temperature became normal, and the rest of the fluid resolved rapidly. In a few weeks no physical signs were left beyond a dulness at the base behind.
11. A child of 5, with a small empyema, following pneumonia of the left base, recovered after 2–3 ounces of pus had been removed by the aspirator.
12. A man of 29, with left-sided empyema, made rapid recovery after 10 ounces had beeu removed.
13. A man of 32, with an empyema of the right side of eleven weeks' duration, refused to have his side opened, and was accordingly tapped only, with the removal of 10 ounces of pus. Sevcral punctures were made subsequently to see if any more pus was present, and none could be found. Recovery was rapid.
14. A man of 19 was tapped five weeks after the commencement of his illness, and 45 ounces of pus removed. Recovery was rapid and uninterrupted.
15. A child of 3 developed a small empyema on the left side after whooping-cough. Four ounces of pus were removed with the aspirator, and recovery was complete.
16. A man of 45 was found to have an empyema on the left side, which had been preceded by pneumonia on that side three months before. After being under observation for a short time, he developed paraplegia, which rapidly became complete. The fluid increasing in the chest, paracentesis was performed and 40 ounces removed. Forty days later a second paracentesis removed 20 ounces. Ultimately the patient got quite well of both paraplegia and empyema.
17. A girl of 4, with left-sided empyema, was tapped twice, 16 ounces being removed on the first occasion and 12 on the second, four days later. Recovery was rapid.
18. A child of 10 months was tapped and pus removed from the left side one month before she was seen. She required no further treatment, and in a month was well.

In the above cases paracentesis alone was sufficient to effect cure, and no pus was expectorated. In the following case, towards the end, a small amount of pus was also coughed up.

19. A man of 41 was admitted with an empyema of the right side of recent development. The temperature was very irregular, hectic it might be called, but it hardly rose at its maximum above normal. Sixty-two ounces were removed on the first paracentesis, and, fourteen days later, 15 ounces. In seven weeks the patient appeared to be perfectly well, although there was still some dulness at the base behind. A month later some fresh pain appeared, and three weeks later, *i.e.*, ten weeks after the second paracentesis, the side was aspirated again and 24 ounces of pus removed. This was followed by the expectoration of a little pus daily, but it rapidly ceased, and recovery was complete.

This case forms the link between those cured by paracentesis and those which spontaneously healed after expectoration, the course which, before paracentesis and incision were so successful, used to be regarded as the most favourable an empyema could take.

Ringer[1] records 2 cases: a child of 2 years old, in which two paracenteses, with the removal of 16 and 6 ounces respectively, were followed by cure; and another, a girl of 4½, in which a single paracentesis, with removal of 19 ounces, led to rapid recovery.

Broadbent[2] and Cheadle record a case of double empyema. The patient, a woman of 31, pregnant with her fifth child, had double empyema. The right side was tapped and 25 ounces withdrawn, the next day the left, with the evacuation of 18 ounces. Five days later the right was tapped again, and 22 ounces more withdrawn, after which the patient made a rapid recovery.

Sangster[3] also records the case of a child of 2 years of age in which the right side was incised and the left tapped twice, 2 and 6 ounces respectively being removed, when the patient got quite well.

Netter records 10 cases of empyema following pneumonia in the adult, all of them treated by paracentesis, and all of them followed by cure. He goes so far as to advocate paracentesis rather than incision in the adult for the empyemata which follow pneumonia, and he gives a few cases of similar success with streptococcus empyema.

In many cases in which paracentesis has been performed, the track of the puncture suppurates. This is, no doubt, due partly to infection of the wound from the pleura, and partly to the pus being mechanically forced, by coughing, from the pleura into the wound. To avoid the latter risk it would be well to strap the side firmly with pads placed over the puncture for a day or two after paracentesis. Do what we will, suppuration of the puncture often occurs, the empyema points through the puncture, and incision becomes necessary, or, if left alone, spontaneous discharge occurs.

Granting that paracentesis may suffice to cure an empyema, it is necessary to consider only the conditions under which it would be right to attempt this method.

In the first place, paracentesis can do no harm, even if an ultimate incision becomes necessary; indeed, it may often do good, for it gains time, and enables the patient to be brought into a better condition to bear the operation, so that as a preliminary to incision it is of the greatest possible use. Being sometimes of itself sufficient to effect cure, there can be no harm in at any rate trying the effect of one, and perhaps a second, paracentesis before proceeding to incision.

In one series of 37 cases, paracentesis was performed prior to incision in 23, and 4 were cured by paracentesis only. In a second series of 119 cases, paracentesis was performed prior to incision in 51, and 10 were cured by paracentesis only.

With encysted or localised empyema it seems to matter little whether an incision be made or not, either in the child or in the adult. Perhaps on the whole a free incision in such cases is the most expeditious means of cure; but where the empyema is deep-seated and the pus difficult to reach, it would be certainly well to try paracentesis first.

It is not, however, always easy to be sure that we have an encysted empyema to deal with. It may be a small empyema in the general cavity, and not shut off by adhesions or encysted.

[1] *Practitioner*, xi. [2] *Med. Press and Circ.*, 1878, ii. [3] *Lancet*, 1880, ii.

In the adult the objections felt to laying the side freely open are greater than in the child, and rest partly on theory and partly on clinical experience.

The theoretical objections are based upon the assumption that, when the side is opened, the lung becomes completely collapsed, so that a very large cavity is necessarily left, which can only heal by granulation from the bottom.

This theory is, however, misleading, for in many cases the lung expands at once, and the cavity becomes rapidly diminished in size; and this may happen even when the lung has been long compressed by fluid, and the empyema been of many weeks', or even months', duration.

The objections based upon experience are more serious; for although there are many cases in the adult in which, even in chronic cases of empyema, cure is rapidly effected after free incision, still there are many others in which the lung does not expand as we should expect, and in which the patient is left with a large cavity, from which a free discharge takes place, and may continue for months or years; yet I do not know that these cases are likely to do any better if treated by paracentesis, for in many of them the lung is permanently diseased and often tubercular.

It is impossible to lay down any general rules for the decision as between paracentesis and free incision. Each case must be dealt with on its own merits; but it is something to know that the free incision is not imperative, and that paracentesis sometimes is sufficient to effect a cure. Whether this will be sufficient in any given case or not, no one can tell until it has been tried. Careful paracentesis can do no harm, nor will mischief be done by postponing free incision for a little while, though I think it ought not to be postponed for long. It might be a good rule of practice, in the adult at any rate, to try the effect of paracentesis first, and if the fluid re-accumulate, and no improvement take place after, say, a second or at the most a third paracentesis, in the course of two or three weeks at the outside, to proceed to incision. At the same time I cannot say that I adopt any general rule of practice in my own cases. Special circumstances, and especially the general condition of the patient, may make me decide at once upon free incision in one case, and in another to try the effect of paracentesis.

If paracentesis be performed for empyema, the aspirator should, if employed at all, be used with great care and without forcible suction, for the lung is often brittle in places, and is likely to be ruptured; and it is a most undesirable thing to burst the lung, and thus convert an ordinary empyema into a pyo-pneumothorax.

If the pus will not flow readily through the aspirator, rather than employ much suction it will be better to stop paracentesis, and proceed later to incision, or else, perhaps, attempt to remove the pus by the simultaneous introduction of air; that is, by Perflation, as it has been called.

Perflation.—There are some cases of empyema in which, though there be a large effusion, only a small amount of pus can be removed.

Thus, for instance, in a case of Mr. Parker's, where incision showed that the side contained 40 or 50 ounces of pus, only 4 could be obtained by means of the aspirator. The explanation often given of these cases is that the chest walls are rigid and the mediastinum and heart fixed, so that when the aspirator is used, contraction cannot take place in the side and the pus will not flow. No doubt this is a condition which is occasionally met with, but I believe it to be comparatively rare. It was for such cases as these that perflation was suggested.

Perflation consists in the simultaneous introduction of air and the withdrawal of pus. It requires two punctures, one in the upper part, by which the air is introduced, and the other in the lower part, by which the fluid is withdrawn.

The air which is introduced must be carefully sterilised or rendered aseptic. This was attempted in some cases by passing the air through water containing antiseptic substances; but it is more satisfactory to filter it through cotton-wool saturated with the disinfectant.

Mr. Parker's name is connected most in this country with Perflation, and his apparatus is figured in the *British Medical Journal* for 1883. The method is ingenious, but cumbersome, applicable to but very few cases, and to these free incision would probably be more suitable, so that I do not think this plan is at all likely to come into any general use.

It is interesting in this association to remember that Hippocrates, who introduced the treatment of empyema by incision, advocated the introduction, at the side of the trocar, of a bladder which could be blown up from the outside so as to force the pus out through the trocar, and thus completely empty the pleura.

64. TUBERCULAR PLEURISY.

Of the part which tubercle plays in the causation of pleurisy, much has been already said in the preceding section. Here it will be necessary to deal with the subject only in its general aspects.

Tubercular pleurisy is sometimes part of a general miliary tuberculosis; it may occasionally be primary; but it is generally a secondary affection, consequent on tubercle of the lung, of the bronchial or mediastinal glands, of the bones of the thorax, of the peritoneum, or other organs adjacent to it.

As a part of general tuberculosis, pleurisy falls into a subordinate position in comparison with the predominant affection of other organs, *e.g.*, the lungs or the meninges of the brain.

When pleurisy occurs in the course of manifest phthisis, it is merely a complication, and its importance will depend upon the form that it takes.

It is rather in those cases where there is no manifest disease in the lung, nor any obvious connection with tubercular mischief in other parts of the body, that the diagnosis becomes of importance as well as of difficulty.

MORBID ANATOMY.—The lesions are best studied where the pleurisy is secondary to tubercle in the lung. The lymphatic tissue beneath the pleura is the favourite seat for these secondary tubercles. They are often very minute, for some time discrete, and in the early stages surrounded by a zone of hyperæmic blood vessels.

The pleura over them becomes covered with inflammatory exudation, and this may rapidly lead to the formation of fibrous adhesions, with obliteration of the pleural cavity. These adhesions are, for the most part, free themselves from tubercles, but they may contain many and even conglomerations of them, forming large caseous masses surrounded by connective tissue.

In most cases, in the earlier stages, the inflammation is associated with effusion, serous, sero-purulent, purulent, or hæmorrhagic, as the case may be; while if there be a tubercular cavity near the surface of the lung and this rupture into the pleura, empyema or pneumothorax, with or without effusion, will be the result.

The chance of a given pleurisy being of tubercular origin varies with the form of the disease. It is least with empyema, of which not more than about 10 per cent. can be attributed to tubercle. It is much greater with serous effusions and with dry pleurisy, but how frequent it is hard to say. Of 101 cases examined *post-mortem* for this purpose by Osler,[1] about $\frac{1}{3}$, that is 32, proved to be tubercular. In the rest no tubercular evidence was forthcoming. These statistics are lower than those given by many other writers, and there is a growing opinion that these forms of pleurisy are more frequently of tubercular origin than used to be supposed; at the same time, it is not necessary to go so far as some authors do, and believe that the great majority of pleurisies, apparently simple in origin, are really of tubercular nature.

[1] *Tubercular Pleurisy.*

DIAGNOSIS.—The diagnosis during life is by no means easy; for, on the one hand, in a certain number of cases, in which there is every reason to believe that the pleurisy is of a simple character, tuberculosis develops subsequently; while on the other hand in many others in which a tubercular origin of the pleurisy is strongly suspected at the time, no further evidence of tubercle develops, and recovery is apparently complete. At the same time, with reference to these cases, it must be borne in mind that pleurisies, which are undoubtedly tubercular, may become quiescent and the disease be arrested.

The diagnosis is, as a rule, easy when pleurisy occurs in a patient who presents obvious signs of tubercle elsewhere, especially in connection with the organs of the chest. It is, of course, true that pleurisy, excited by a tubercular affection of an organ near, is not necessarily itself tubercular, but, from a clinical point of view, this is a distinction without a difference; for, whether the resulting inflammation itself become tubercular or not, it has been excited by tubercle, and this, from a clinical point of view, is practically the same thing, so that these cases are rightly described as "Tubercular Pleurisy."

Where there are no obvious signs of tubercle in other organs, the diagnosis may prove very difficult.

The mode of onset may be of little value, for a tubercular pleurisy may be acute or chronic, insidious or latent. On the whole, it may be said that the more gradual and insidious the onset and course, the more likely is the pleurisy to be of tubercular nature.

Nor is the family history of much assistance, except in so far as it shows a diminished vitality and lessened resistance to the attack of the tubercle bacillus.

Indeed, in the majority of cases the diagnosis rests chiefly upon the character of the inflammation and upon the clinical course it takes.

The inflammation may be of any character, either dry or with effusion.

When the effusion is dry, its tubercular nature may be suggested by peculiarities in its seat or extent.

The common seat of simple dry pleurisy is in the mid-axilla, or at the base of the lung, that is to say, many of the cases which occur in this locality are not tubercular in origin.

If dry pleurisy is found at the apex, it is almost certain to depend upon the common lesion in that part of the lung, viz., tubercle.

Again, if it be of unusual extent, involving the greater part, or even the whole of one side, it will probably depend upon a widespread lesion, and that is most likely to be tubercle.

Again, if it be bilateral, especially if it involve both apices, it will be almost certainly the result of a bilateral lesion, and the commonest of them by far is tubercle.

When effusions form, the fluid may be serous, sero-fibrinous, sero-purulent, purulent, or hæmorrhagic.

Empyema may be put aside as being, on the whole, rarely of tubercular origin. For even when it occurs in the course of phthisis, it is often of strepto-coccal and not tubercular origin. The other effusions are much more frequently tubercular, and the probability will be increased if the fluid be blood-stained or turbid, but their characters are only suggestive, not conclusive.

Bacteriological investigation often fails to throw any light on the question. In the majority of cases serous effusions are sterile, that is to say, no bacilli can be discovered by the microscope or cultivated in the usual media. Occasionally

fluids, which otherwise seem to be sterile, can be proved to be tubercular by the results of inoculation into animals; but, as a rule, inoculation also fails. At the same time, these facts are true also of pleurisies which are undoubtedly tubercular, so that negative results of this kind do not prove the effusion to be non-tubercular. Injections of tuberculin have been also used for the purposes of diagnosis, but the results are not reliable, and even if the reaction be obtained, it does not necessarily follow that it is in the pleura that the tubercular disease is seated.

It is generally by the subsequent clinical history of the case only that the diagnosis can be determined; for it not infrequently happens that cases which raised no suspicion of tuberculosis at the time develop signs ere long in apparently direct connection with the original pleurisy. On the other hand, it does not follow that because no signs of tubercle develop subsequently, the original pleurisy was of simple nature; for, even in undoubtedly tubercular cases, the disease may become quiescent and no further mischief develop.

I think in the case of pleurisy, as of many other diseases, it is wise always to suspect the nature of a case which presents eccentricities. Thus it is that hæmorrhagic and turbid effusions are suspicious. I believe, also, though many authorities do not hold this view, that spontaneous coagulation of the fluid which has been removed by paracentesis is very suggestive too. Again, I have seen several cases in which unusually rapid recovery from an apparently simple pleurisy was followed by tuberculosis.

Thus I remember a young woman, aged 25, with a previously healthy history, who was attacked with acute pleurisy. There was nothing remarkable in the case, except that an effusion formed rapidly, and soon required tapping, and on removal the fluid spontaneously clotted. After paracentesis the effusion did not return.

Convalescence was extremely rapid, and in a few days the patient appeared to be quite well. However, about eight weeks after her illness she spat up a little blood, quickly developed signs of pulmonary tuberculosis, and died before long of acute phthisis.

CLINICAL TYPES.—Tuberculosis of the pleura presents itself, like tuberculosis of the lungs and other organs, in the three clinical forms, viz., the acute, subacute, and chronic.

I. Acute Tubercular Pleurisy.—We do not know the proportion of acute pleurisies which are really due to tubercle, for most of the acute cases get well, and some of these are certainly of tubercular origin.

A. In one class the onset is acute, but the acute symptoms subside, the condition becomes chronic, ultimately the patients die, and tubercular disease of the pleura is found.

B. In another class the patients are already obviously tubercular, suffering either from phthisis or tubercular disease elsewhere. Soon pleurisy sets in and plays an important part in the fatal result.

C. The third class is that in which the inflammation is of a suppurative character. Empyema is of a tubercular origin only in a comparatively small number of cases, and in these is not usually associated with very acute symptoms, so that an empyema of very acute onset is most likely not to be tubercular, yet some tubercular empyemata are very acute both in onset and course.

II. Subacute or Chronic Tubercular Pleurisy.—This may lead to serous effusion, to purulent effusion, or be of the dry, adhesive form, and result in more or less extensive obliteration.

A. With serous effusion.—These cases fall into two groups: first, those in which the effusion occurs in patients who are already the subjects of phthisis; secondly, those in which it is apparently primary, *i.e.*, occurs in patients who are

not already tubercular, so far as is known. This latter group has of recent years greatly enlarged its borders, for an ever-increasing number of them is shown by their subsequent course to have been of tubercular origin.

These effusions usually have a greenish tint, and occasionally are a little turbid. They are not so frequently hæmorrhagic as are those of more acute onset, but, like them, they are usually sterile.

B. With purulent effusions.—Empyemata of tubercular origin may, as has been stated, be sometimes acute, but they are usually chronic and sometimes latent. The effusion is rarely pure pus, but of a sero-purulent character, somewhat tinged with blood, and containing much fatty matter. Empyemata, even when developing in phthisical persons, do not necessarily contain the tubercle bacillus, but more often the streptococcus or staphylococcus.

C. Chronic adhesive tubercular pleurisy.—This is generally associated with a considerable thickening of the serous membrane.

The commonest form is that met with in phthisis, where there is great thickening of the pleura at the apex, though in some of these cases the lower part of the pleural cavity is occupied by a localised serous effusion.

In other cases the pleurisy is, from the onset, dry, *i.e.*, not associated with effusion at all, but immediately leads to adhesion. The inflammation is of a proliferative character, and attended with considerable thickening as well as induration, so that the new-formed tissue may measure half an inch in thickness or more.

In some of these forms the tubercular inflammation may spread into the lung along the dissepiments. It is this form of phthisis which Charcot described as chronic "pleurogenic phthisis," *i.e.*, chronic tubercular disease of the lung of pleural origin.

This is the place to refer to an affection upon which the late Sir Andrew Clarke wrote, under the title of "primitive dry pleurisy." He described the disease as running through four stages: first, the neoplastic pleural membrane stage, *i.e.*, the stage of ordinary dry pleurisy; secondly, the fibroid-lung stage; thirdly, the bronchiectatic stage; and lastly, the phthisical fibroid-lung stage. As in the full development of the disease the case ends in tubercular phthisis, most people would regard the condition as tubercular from the onset, differing only from other cases in the chronic course the disease has run.

Clinically the cases are not marked by any conditions by which they can be clearly recognised during life.

Indeed, Sir Andrew Clarke summed up the diagnosis much in this way. If the signs of dry pleurisy appear in the lower part of the pleura, and if there exist no sign of local disease and no serious symptoms of constitutional disturbance, it is presumable that the case is one of primitive dry pleurisy.

Yet we know that tubercular pleurisy may arise exactly under these conditions, and run exactly the course described.

Although the possible existence of a primitive dry pleurisy, such as is contended for, cannot be denied, still it must be admitted that the evidence, both pathological and clinical, brought forward to prove its existence is not conclusive.

III. General Serous Membrane Tuberculosis.—There remains one other group of cases in which tubercular disease of the pleura is associated with similar disease in the other adjacent serous membranes, frequently in the peritoneum.

The names *Poly-serositis* or *Poly-orrhomenitis* have been recently suggested for the group, but it is a pity to invent new names for a familiar condition, which has been long recognised and is not new.

The disease is of subacute or chronic character, and is thereby distinguished from the acute tubercular affection of the serous membranes, which forms so striking a part of general tuberculosis.

Although the pericardium may be involved in these cases, still it generally escapes, so that the stress of the disease falls upon the pleura and peritoneum. Reference has frequently been made to the ease with which infection may pass through the diaphragm from the one cavity to the other, owing to the free communication between the lymphatics on either side. It is remarkable that this secondary infection of the one serous membrane from the other does not more frequently occur.

The cases run a very chronic course, and the peritoneum is usually the first to be attacked.

Thus of 21 cases recorded by Vierordt, the peritoneum was the first to be involved in 13, and the pleura the first in 8.

The general course which the case runs is usually this:—The patient comes under observation with the ordinary signs of a tubercular peritonitis, usually somewhat chronic in character, and probably associated with effusion.

Subsequently either chronic pleurisy develops, or perhaps a subacute or chronic pleural effusion forms, which may require repeated paracentesis; sometimes the patient comes under observation with a considerable effusion both in the pleura and in the peritoneum. In the course of time the mischief subsides, the effusions disappear, and the patients recover, though left, of course, with more or less adhesion, and with the discomforts consequent on it.

They often remain in fairly good health, and do not develop any further signs of tubercular mischief; in other words, as has been already stated, tubercular disease of the serous membranes often remains a purely local mischief.

TREATMENT.—The presence of tubercle may, to some extent, modify the prognosis in any given case, but does not materially affect the general lines of treatment. This only, perhaps, may be said, that in cases of empyema more caution must be exercised before the pleura is laid freely open, for, as experience has shown, the cases of empyema associated with tuberculosis do not do so well as others after the side has been opened; but in many cases there is little choice in the matter, and the presence of tubercle thus affects the prognosis, rather than our treatment.

65. DIAPHRAGMATIC PLEURISY.

Diaphragmatic pleurisy is only a pleurisy in a peculiar place, and with peculiar symptoms, because it involves the diaphragm.

The diaphragm is but a thin sheet of muscle, the lymphatics of which communicate freely with the serous membranes on either side of it. Any inflammation on either side will involve the muscle, and may spread through the diaphragm from the one side to the other.

Of course, where there is a general pleurisy, the diaphragmatic part of the pleura is involved also in the inflammation, but this is not described as "Diaphragmatic Pleurisy," the term being confined to those cases in which the pleurisy attacks the diaphragmatic surface alone or chiefly.

In the great majority of cases, diaphragmatic pleurisy is secondary to inflammation of the peritoneal surface; thus it often follows subphrenic abscess, suppuration in the liver, or other less acute inflammatory affections close to the

diaphragm. Even of cases which appear to be primary, many are really secondary and the result of inflammatory mischief in the base of the lung, *e.g.*, deep-seated pneumonia.

Diaphragmatic pleurisy may be either dry, or with effusion, serous or purulent.

THE PHYSICAL SIGNS.—Besides the general signs of pleurisy, which vary somewhat with the form of pleurisy present, there will be the special signs which depend upon the affection of the diaphragm or of its nerves.

1. *The muscle.*—In the early stages the inflammation irritates the muscle, and causes spasm or contraction in it. In the later stages the nutrition of the muscle will be seriously affected, so that the diaphragm becomes weak or even actually paralysed in the affected part.

2. *The nerves.*—Although the phrenic is not a sensory nerve, the diaphragm is not insensitive. Considerable pain is felt with diaphragmatic pleurisy, just as in other forms of pleurisy; it is of the same character, viz., sharp and stabbing, and is increased on respiration or on pressure. The pain is often referred to special spots, especially to the epigastrium and to the antero-lateral portion of the costal arch.

Besides the pain, there are tender spots, which are the same as those described in connection with neuritis of the phrenic nerve.

i. Almost in the middle of the epigastrium (the epigastric).
ii. Along the costal arch in the antero-lateral region.
iii. Along the edge of the sternum, from the level of the third rib upward.
iv. Between the divisions of the sterno-mastoid at the root of the neck.
v. Over the spines of the middle cervical vertebræ, from the second to the fifth, *i.e.*, over those sections of the cord from which the roots of the brachial and cervical plexuses arise.
vi. Occasionally over branches of the brachial and cervical plexuses, for instance, in the neck or over the shoulder.

These painful points are rather of interest than importance in diagnosis, and, except the two first, are usually absent.

As a result of the pain and of the affection of the muscle, the movements of the diaphragm on the affected side are more or less restricted, and may be entirely absent. In addition to this, the lower ribs are drawn in and down, and fixed by the abdominal muscles, which are somewhat tense and rigid. The respiration thus becomes markedly costal, especially on the affected side. The hypochondrium on the affected side is usually flat, *i.e.*, neither distended nor retracted, but if there be effusion it may be prominent; on the other hand, it may be retracted, and accompanied with inspiratory recession.

The physical signs are often indefinite, especially with the dry form. Friction is often not audible, and unless there be effusion, there need be no alteration in the lines of percussion dulness.

THE SYMPTOMS.—The fever and general illness are much as in other cases of acute pleuritis. The pulse may be rapid and the temperature high, conditions, however, which vary greatly with the amount of pain. Often there is great anxiety and distress, in part the result of pain, and in part such as is met with in most acute diseases of other vital parts, *e.g.*, of the heart or pericardium.

Occasionally there is delirium, but this probably is due to some complication, especially pneumonia.

The pain, referred as stated to the epigastrium, or to the middle of the costal arch, is often intense, especially in the early stage, but diminishes or passes off when effusion forms. To relieve the pain, as well as the dyspnœa with which it is accompanied, the patient may assume the sitting posture, so as to throw the weight of the abdominal organs off the diaphragm, and in order to further check

respiratory movements, may pinch or press in the lower ribs, or find great relief when a bandage is firmly applied round them.

The breathing, and, for the same reason, speech also, is short, jerky, and painful.

Cough, if present, is dry, short, hacking, and may be very painful, but fortunately it is often slight, or even absent, and there is usually no expectoration.

Hiccough is not uncommon, and a most painful complication it is. It may be almost constant night and day, and hardly to be controlled by drugs, even considerable doses of morphia failing to relieve it much. As in abdominal affections, so in diaphragmatic pleurisy, hiccough is not only a troublesome symptom, inasmuch as it greatly increases the distress and robs the patient of sleep, but it is a sign of serious import.

Vomiting also may be troublesome, but fortunately it is rare, and appears to be generally associated with peritonitis.

Jaundice also is described as an occasional complication, but it is accidental, and probably depends in most cases upon the presence of some affection of the liver or upon pneumonia.

The chief complication occurs when the pleurisy spreads through the diaphragm to the peritoneum. Then local and occasionally even general peritonitis may be excited. Of this one of the earliest symptoms is tympanites, due to the dilatation sometimes of the stomach alone, and sometimes of all the intestines.

The symptoms are most severe with dry pleurisy, and it is in these that there are the fewest physical signs or evidences of the disease. With effusion which is generally suppurative, though the general signs may be clearly those of suppuration, it may be by no means easy to determine where the pus is; or, if its seat be indicated, to decide whether it be above or below the diaphragm.

DIAGNOSIS.—In those cases in which pain is the prominent symptom, the diagnosis will have to be made chiefly from pneumonia, from pericarditis, and from peritonitis; from rheumatic affection of the muscles, from neuralgia and spasm of the diaphragm. The diagnosis must be made by exclusion, and may be difficult or impossible.

In those cases in which purulent effusion is present, the diagnosis will have to be made from abscess below the diaphragm, in connection, for instance, with gastric and duodenal ulcers; or from abscess or suppurating hydatids in the liver; or from suppuration due to other causes.

RESULTS.—These depend chiefly upon the extent to which the muscle is affected. Adhesions are almost certain to form with the lung above; and if the inflammation have spread through the diaphragm, also with the parts below it. If the adhesions be with the lung only, there need be no evident effect. If the muscle be much involved, as where the inflammation has spread completely through the diaphragm, a good deal of permanent respiratory defect will remain. If there be adhesions above and below the diaphragm, the muscle will be practically useless on that side; and if adhesions occur with the costal pleura as well, the lower ribs will be much retracted and, if not quite immovable, will show marked inspiratory recession.

With diaphragmatic pleurisy, past or present, pulmonary complications, of course, become grave.

TREATMENT.—The treatment of diaphragmatic pleurisy does not differ materially from that of other forms of pleurisy. If pus be present, it should be evacuated as soon as it is found, but to find it is often a matter of very great difficulty. Considerable knowledge and experience are often necessary to explore

these cases with success. A long needle is often required, and it may have to be introduced several inches into the side. Incision should never be performed until the pus has been previously discovered by the needle. When once discovered, the needle should be used as a director, and the cavity laid freely open and drained.

In some cases, do what we will, though the presence of pus is correctly diagnosed, it cannot be found with the needle. In such cases it is better left alone than groped for with the knife. Fortunately, in most instances, rupture takes place through the lung, and the empyema spontaneously heals.

66. DOUBLE PLEURISY.

The importance of double pleurisy lies in the fact that, whether with or without effusion, it is almost without exception secondary to some lesion present on both sides of the chest.

The commonest of these is tubercle, and the next pneumonia, but, of course, new-growth cannot be excluded. Occasionally it follows general peritonitis, when the inflammation has attacked the under surfaces of the diaphragm on both sides; or it may be a part of some general septic infection.

If it affect both apices, it is with little doubt tubercular in origin. If it affect both bases it may be tubercular, and this would be still further confirmed if the effusion were serous, though this might be the result of new-growth. If there be double empyema, the cause is probably double pneumonia or some septic infection.

Double pleurisy is in all cases serious: first, because of the gravity of the symptoms if it be severe; secondly, because of the causes to which it may be due; and lastly, because of the interference with the respiratory powers it may leave behind.

67. SYMPHYSIS PLEURÆ (Pleuritic Adhesion).

With few exceptions, pleuritic adhesion is the result of past inflammation, and a good deal has already been said incidentally of this in discussing the results of the different forms of pleuritis. Thus it may follow empyema, serous effusion or dry pleurisy, but in the most extreme forms it is the result of empyema.

Occasionally pleural adhesions are found without anything in the history to explain their presence. Some of these cases may be referred to pneumonia or pleurisy in early childhood, the nature of the illness having been unrecognised or the occurrence forgotten.

Thus I saw some time ago an adult in whom the heart was beating under the right nipple. This case had been diagnosed as one of transposition of viscera, yet careful examination showed that this could not be the case, and enquiry ascertained the fact that as a child the patient had been under treatment at the Children's Hospital, for what I have little doubt was pleuritic effusion, so that the heart had remained in the position to which it had been displaced at the time of the effusion.

In the great majority of cases, where no history is forthcoming, the cause of pleural adhesion is tubercle. Thus, out of 69 cases examined for this purpose by Ziemssen,[1] evidence of tubercle, either in the patient at the time, or in the history of the patient or of his family, was obtained in one-third of the cases.

[1] *Festsch. Virchow*, 1891, iii. 274.

Sometimes, without any history of definite illness, the patient on enquiry will confess to recurrent attacks of slight pain from time to time in different parts of the chest, and these are almost invariably the result of recurrent pleurisy of tubercular origin.

Fig. 155.—Chronic pleuritic thickening, a little reduced from its natural size. It measured about three-quarters of an inch in thickness.

The **morbid anatomy** of the condition is simple. The adhesions are formed of connective tissue which often contain new blood vessels. If of tubercular origin, they may also contain tubercles, but not necessarily. Usually there is thickening of the pleura, more or less, both on the costal and pulmonary side. In some cases this may be so slight as to produce no evidence by which it could be recognised during life. In other cases it may be considerable, so that the pleura, in the affected part, may measure half-an-inch or more in thickness.

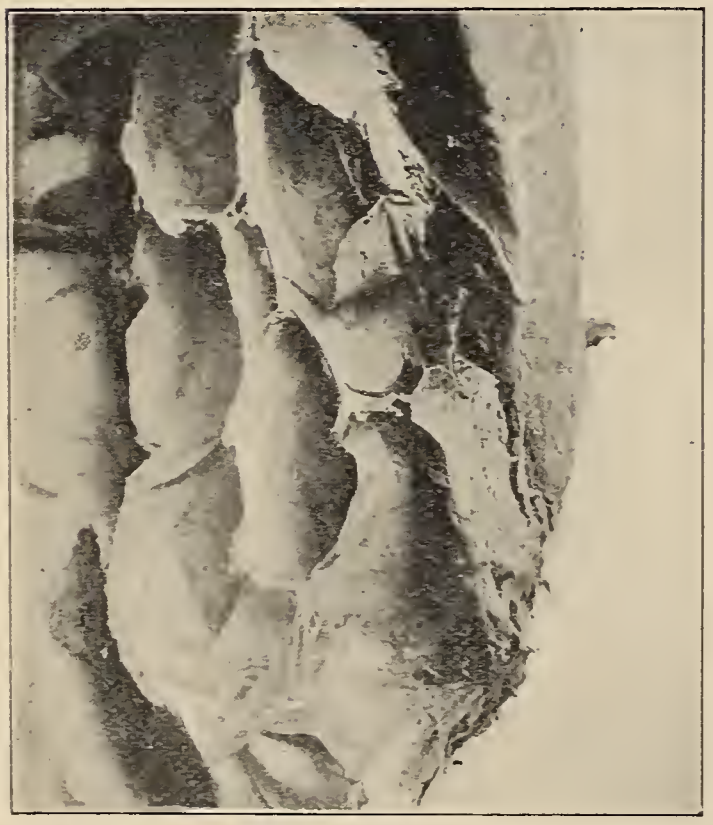


Fig. 156.—Areolar pleurisy (Museum, Royal College of Surgeons). The pleural cavity is divided into irregular spaces by membranous adhesions; the loculi were filled with serous fluid; the lung is seen posteriorly, and the loculi have been opened from the front.

As to the nature and cause, the seat of the adhesions may give some indication; thus at the apex, the adhesions will almost certainly be of tubercular origin, and associated with tubercular changes of a more or less chronic character in the lung beneath; at the base, though they may be tubercular, they are more likely to have some other origin, and to be the result of past inflammation, with or without effusion.

When effusions are present, and the lung more or less collapsed, the adhesions may be drawn out into long bands, and if the effusion be chronic, these bands may become covered with flattened epithelial cells on either side, similar to those covering the rest of the pleura.

The most peculiar form of pleural adhesion is that in which loculi or spaces of irregular shape and size are formed, which contain fluid, sometimes of different nature in the different loculi (*cf.* Loculated Empyema).

Such adhesions may snap, and the patient be even conscious of the occurrence by the sudden pain which is felt.

When they snap, hæmorrhage may take place from the ruptured vessels, and this is the explanation, no doubt, of some of the cases of hæmorrhagic effusion in simple pleurisy.

Where the adhesions have been of simple inflammatory origin, they may, after a time, disappear to a great extent, or sometimes entirely, so that the pleura may return to its normal condition, and no trace of the old inflammation be left. This is, however, not the rule; usually they persist, and leave more or less permanent interference with the movements of the lung.

The result of pleural adhesion is, of course, some interference with the respiratory movements of the lung. If, however, the adhesions be thin, the defect in the pleura may be compensated for by the elasticity of the neighbouring parts of the lung, and no signs of the condition will be apparent.

If the adhesions are denser, contraction takes place in the new-formed connective tissue, and the affected part of the chest becomes flattened. The impaired movements diminish the free entry of air into that part of the lung, and in consequence the respiratory murmur will be diminished, while, if there be much thickening, the percussion may be impaired, and the vocal vibrations and vocal resonance diminished or even absent. The physical signs are most evident when the pleural adhesions are at the base of the thorax, for there it is that the respiratory excursion is largest.

The **only symptom** likely to be produced is shortness of breath, especially on exertion, but this is not likely to be marked unless the adhesions are considerable. The importance of the adhesions, even when not sufficient to produce any symptoms or physical signs under ordinary conditions, is seen when any pulmonary complications develop, such as pneumonia or bronchitis; then dyspnœa is often experienced out of all proportion to that apparently required by the disease, and in not a few of the fatal cases of pneumonia, unsuspected pleural adhesions explain the gravity of the symptoms and the fatal result.

According as the adhesions form between the lung and the ribs on the one hand, or the diaphragm on the other, the pleural adhesions are described as *Costo-pulmonary* or *Phreno-pulmonary*; the former is the most common, the latter the most important.

In the phreno-pulmonary form, the diaphragm and lung are closely adherent to one another, and when, as usually happens, the costal pleura is also involved, the lower part of the chest becomes greatly flattened, retracted, and almost immovable, the movements of the diaphragm are much impaired, and may be entirely absent on the affected side, while in some instances there is a perversion of respiratory type, so that inspiratory recession occurs. In extreme cases of this kind, the muscular tissue of the diaphragm may atrophy and almost entirely disappear, as the result, not of the disuse only, but of the interstitial cirrhotic change which follows.

The **prognosis** of pleural adhesions depends chiefly upon the cause, and upon the extent.

Where the adhesions are of tubercular origin, they will probably be permanent and possibly progressive. Where the adhesions are of simple inflammatory origin, and of slight extent, they may in time entirely disappear, or, at any rate, produce no symptoms. Even when they are extensive, it is extraordinary what time and nature will do towards recovery, and in case of extreme deformity, such as follows empyemata in children, recovery may in the end be complete, and the symmetry of the chest be restored to such an extent that it may be difficult after a time even to tell which side it was that had been affected.

Treatment.—In all cases, in addition to general treatment, recovery may be greatly assisted by appropriate physical exercises and pulmonary gymnastics.

68. HYDROTHORAX, HYDROPS PLEURÆ, DROPSY OF THE PLEURA.

Hydrops pleuræ has to be distinguished from inflammatory effusion into the pleura. The effusions have been called transudation and exudation respectively. Both are serous fluids, but the inflammatory effusion contains more or less fibrin as well as some red and white cells, from which dropsical effusions are almost, if not quite, free.

The surface of the membrane is smooth, and there are no signs of inflammation; the sub-pleural tissue may be œdematous, and so may the lungs, but the parts of the lung near the fluid are generally more or less compressed and collapsed.

Hydrops pleuræ is, with few exceptions, bilateral; when unilateral, there is generally some local cause for it; either on the affected side, as, for instance, the presence of a tumour, or on the opposite side, which may be obliterated by old adhesions. Where past pleuritic inflammation has led to partial adhesions, the dropsical effusion may be localised or even loculated, but these are very rare conditions.

The quantity of fluid varies; it cannot be of large amount unless it happens to be unilateral, for obvious reasons. The amount is rarely equal on the two sides, that on the right being usually the larger. The difference is determined chiefly by position, the effusion being larger on that side upon which the patient habitually lies.

In one case, in the course of tubal nephritis, the effusion formed at the rate of $3\frac{1}{2}$ ounces per hour for about six days, calculated by the amounts removed by paracentesis. On death the large azygos vein was found thrombosed, the effusion being unilateral.

Dropsical effusions may form with considerable rapidity, but they are rarely absorbed at anything like the same rate.

The small quantities, which are found in almost every autopsy, have occurred just before death, and are of no significance.

Hydrothorax is usually a part of general dropsy, and one of the last conditions to arise in it. The description often given of such a case is graphic. The feet, it is said, first began to swell, then the legs, and then the stomach, and when the "water reached the chest" the patient died.

The effusion into the pleura may be an early symptom of general dropsy, and sometimes even the first, just as we sometimes see ascites develop early, or the liver become enormously enlarged before there is general dropsy at all. There are in these cases, I suppose, generally some local reasons for the exception to the rule.

The dropsical effusion usually disappears in the reverse order, if the patient recover; thus the effusion in the pleura will be the first to go, just as it was the last to come. However, there are exceptions to this rule, and the pleural effusion may continue even when the dropsy has left all the rest of the body.

Thus I remember a case of cardiac dropsy in which the patient had had effusion into both pleural cavities. In the course of time all signs of dropsy disappeared except from the right pleural cavity, and this remained half filled with fluid. It was not until this had been tapped and the cavity emptied that recovery took place.

The **causes of hydrothorax** fall into two groups, according as the condition is a part of general dropsy or not. In the former case the causes are the same as those of general dropsy, and may be arranged conveniently into the three groups—the cardiac, the renal, and the hæmic.

In the *cardiac group* must be included certain other cases besides those in which the primary disease lies in the heart, such as emphysema notably, and some forms of mediastinal tumour.

The factor common to all these cases is failure of the right side of the heart. As long as this does its work and compensation is complete, no venous congestion will arise; but as soon as it fails, the signs of venous congestion appear and general dropsy develops.

In the *renal group*, the commonest cases are parenchymatous nephritis and amyloid disease. The cause of the dropsy lies probably in nutritive changes in the minute vessels. In granular kidney, although the vessels are diseased over the whole body, still dropsy is rare; and when not due to intercurrent acute nephritis, it is probably cardiac in origin, the result of failure of the hypertrophied left ventricle.

In the *hæmic group* the dropsy is, in many cases no doubt, also of cardiac origin, for even in the slighter degrees of anæmia the heart is weak, and in extreme cases is markedly fatty; but in other cases it is to be attributed probably not so much to the heart as to changes in the nutrition of the small vessels, or to alterations in the blood itself, such as are met with in many of the grave anæmias and cachexias.

When hydrothorax is **unilateral,** there are generally some local conditions of disease to account for it; thus, if the pleural cavity be obliterated by old adhesions on the one side, it is obvious that a hydrothorax can occur on the other side only.

Old adhesions also account for some of the peculiar forms in which hydrothorax may occur, *e.g.*, where it is localised or loculated.

Apart from these conditions, if hydrothorax be unilateral, it is usually associated with some local disease on the affected side of the chest, *e.g.*, new-growth in the mediastinum, pleura, or lung of that side, an aneurysm of the aorta, or, in some rare cases, fibrous bands pinching the large vessels at the root of the lung. Hydrothorax has also been met with where the venæ azygos are plugged or otherwise obstructed, so that the effusion would appear to come from the parietal, *i.e.*, the intercostal, veins.

In many of these cases the effusion is described as inflammatory, but I believe it to be more truly dropsical. It is often associated with obvious compression of the big vessels at the root of the lung, and with obstruction to the circulation through them.

It is not agreed from which set of vessels the effusion comes, but I think it must be from the pulmonary artery or vein; and though the obstruction often leads rather to œdema of the lung than to pleuritic effusion, still the two are frequently associated together. The line between chronic inflammatory effusion and dropsy is, indeed, as difficult to draw in the pleura as it is in the peritoneum.

One condition remains to be spoken of which does not fall into any of the preceding groups, viz., that which is usually called **"Hydrops ex vacuo."** In this condition, as the result generally of past inflammation, the lung is bound down so that it cannot expand; and as the walls of the chest cannot fall in enough to come in contact with the contracted lung, a space is left which is filled with fluid. This condition may be regarded as practically incurable, for as often as the fluid is removed, it re-accumulates. At the same time, the presence of the fluid causes little discomfort unless the amount be very large. It is this class of case which provides the chief instances of "chronic pleuritic effusion" of long duration.

For the cure of such cases nothing obviously can be done, except an Estländer. This operation has, I believe, never been deliberately performed for this condition, but it is conceivable that it might be appropriate for some rare cases. Yet, considering the gravity of the operation and the uncertain result of it, the patient would probably be running less risk if the fluid were left, than if the operation were attempted.

Diagnosis.—The diagnosis is, as a rule, quite easy. There are the physical signs of fluid, with but few exceptions, at both bases, and there is the general dropsy to suggest the cause.

The physical signs are not always quite simple, for on the one hand, if there be a large amount of subcutaneous œdema, the percussion will be difficult, and on the other, pleuritic effusion is not always easy to diagnose from œdema of the lung. In contradistinction to inflammatory effusion, it is stated that in hydrothorax the level of the fluid is more horizontal, and shifts more readily with position; in other words, that Damoiseau's curve is not present; but this curve is not of any importance in the diagnosis of inflammatory effusion, nor can any difference be made out in the readiness with which the fluid shifts its position in the two cases.

Symptoms.—The symptoms are very often indefinite, probably nothing more than gradually increasing dyspnœa, for which, of course, there may be many other causes besides pleural effusion. It is a good routine rule in cases of general dropsy, especially if there be any dyspnœa, to examine the back of the chest carefully and see whether fluid be present.

Occasionally dyspnœa is considerable, so that the patient cannot lie down (orthopnœa); sometimes it is paroxysmal, as the cough, too, may be. In these cases the symptoms are not so much connected with the effusion as with the heart.

One point to be remembered is, that a very small amount of fluid may give rise to a considerable amount of dyspnœa, and, *per contra*, that the removal of even a small amount of fluid may give great relief.

There is, as a rule, no fever, no pain, no friction. Cough, too, may be absent, or, if present, is more likely the result of congestion of the lung than of the pleuritic effusion.

Prognosis.—The general prognosis will depend upon the cause, but as in most cases hydrothorax is a part of general dropsy, and a late event in the course of it, its appearance is of somewhat grave omen.

Treatment.—The general treatment is that of the condition upon which the hydrothorax depends, and in most cases, as stated, it is a part of general dropsy. Thus it has been recommended to use diaphoretics and hot baths, free purgation, and diuretics. The objection to these methods of treatment is that they are exhausting, and produce a good deal of depression. Many of the cases of hydrothorax are of cardiac origin, and these remedies make the cardiac condition rather worse than better; the treatment by stimulants and tonics, such as digitalis and iron, is more effectual, and to these may be added citrate of caffein, one of the most useful remedies of its class, acting, as it does, both upon the heart and upon the kidneys.

The special treatment of hydrothorax is paracentesis, and this should be performed without hesitation whenever there is dyspnœa, even if the amount of fluid be small; for, as stated, a small quantity of fluid may, in the condition in

which it arises, cause a good deal of distress, and the removal of even a small quantity give very great relief.

As stated, when general dropsy is diminishing, the pleura is the first place to show signs of improvement, and the fluid may disappear there quite early in the case. In the same way, if the œdema of the lower part of the body be relieved, the fluid from the pleura may also be absorbed; in other words, the level, as it were, of the fluid may fall. Thus absorption may follow puncture or tapping of the legs where the œdema in the legs is considerable. There are two risks in this treatment. The first is that of infection, for unless the parts be kept very clean, and the capillary tubes used be strictly aseptic, inflammatory processes may be started, which may take on gangrenous action, with very serious results. Under any circumstances, there still remains the second objection, that the removal of such a large quantity of albuminous fluid, often as much as a gallon or more in the twenty-four hours, is attended by considerable exhaustion, even though local relief be given to the chest symptoms; for it is evident that a drain of so much richly albuminous fluid from the body must be a very severe tax upon the powers of the patient, and will greatly increase the general weakness.

69. HÆMORRHAGIC EFFUSION.

Hæmorrhagic effusion must be distinguished from hæmothorax. In the former there is an effusion which is blood-stained, in the latter the fluid consists of pure blood.

Towards the end of paracentesis the fluid may become blood-stained, especially if it has been drawn off too rapidly, or under too great pressure. Those cases in which the presence of blood is accidental are not included.

In hæmorrhagic effusion the fluid, whether serous or purulent, as it may happen to be, is coloured by the admixture of more or less blood. The colour depends upon the amount of blood, and upon the time the blood has been present in the fluid, being of a red colour when recent, and of a dark colour when of some standing.

Purulent effusions are less often hæmorrhagic than serous, and generally contain but little blood, so that they are rarely of more than a salmon-pink or terra-cotta colour.

All serous effusions contain some red blood cells, but in ordinary cases not in sufficient numbers to give the characteristic colour. This does not appear until the red cells number 5000 or 6000 in the cubic millimetre.

The colour is pink when blood is present in small amount only, red when in larger amount and recent, and in other cases it may be dark or even black, if the blood has been present for some time in the fluid.

The presence of blood may be the result of direct exudation from the blood-vessels owing to the intensity of the inflammation, or of the rupture of the small thin-walled vessels in the adhesions, or in the case of cancer from the newly-formed vessels in the growth. In other cases, we may assume the vessels themselves to be diseased, as in granular kidney and atheroma, and probably also in scurvy, hæmophilia, and in hæmorrhagic fevers.

The blood is never in sufficient amount to clot. Some serous effusions coagulate spontaneously, and this they may also do when blood is present, but the clotting does not depend upon the presence of the blood.

The **frequency** with which serous effusions are found to be hæmorrhagic is small, not more than 6 per cent., and it is still smaller with empyema.

The **diagnosis** can only be made by means of a needle, for there is usually nothing to suggest that the effusion is hæmorrhagic. The discovery is usually accidental, and it so far affects the prognosis that it gives evidence either of an intense inflammation or of the presence of some serious complication, such as tubercle or cancer.

The **prognosis** of hæmorrhagic effusion, speaking generally, is worse than that of simple serous effusion, but in most cases it chiefly depends upon the cause. Thus with cancer the prognosis is that of the original disease, as it is also with tubercle. In many of the tubercular cases, however, recovery takes place, at any rate for the time.

The **treatment** of hæmorrhagic effusion does not differ from that of serous effusion.

The chief causes of hæmorrhagic effusion are tubercle and cancer. Besides these, there is a miscellaneous group of cases in which the presence of blood is attributed to conditions such as cirrhosis of the liver, granular kidney, and general hæmorrhagic states.

Speaking generally, if blood be found present in an effusion, the first thought is of tubercle, next of malignant disease, then of some intense simple inflammation, and lastly of one of the rare miscellaneous causes just mentioned.

i. *Tubercular.*—Hæmorrhagic effusion is most likely to be found associated with the acute tubercular processes in the lung or pleura, rather than with the chronic; but it may occur with either, and is sometimes the first evidence given of any tubercular mischief at all.

Sometimes an effusion which has been markedly hæmorrhagic at first becomes less and less so as paracentesis is repeated, and ultimately becomes serous. This happened in a case of Dieulafoy's, in which the fluid was found serous on the seventh paracentesis, and the patient made a good recovery.

The frequency of hæmorrhage with tubercular pleurisy is explained by the congestion which surrounds the tubercles in their early stage, by the vascularity of the new membrane which is formed, by the degeneration which takes place in the small vessels, as well as by the involvement of the vascular walls in the tubercular changes.

It is stated that hæmorrhagic tubercular pleurisy is rich in fibrin, and comparatively poor in red cells, as compared with that in malignant disease; but this probably means only that there is often a freer hæmorrhage with cancer than with tubercle.

ii. *Cancerous.*—The effusion associated with malignant disease is by no means hæmorrhagic in all cases; probably in less than half. When present, the blood is in larger amount, and the effusion is darker in colour. I have removed, from a gentleman dying of cancer, fluid which, though clear, was as dark as port wine.

In these cases the effusion generally re-forms rapidly, and continues to be hæmorrhagic.

The diagnosis is, as a rule, easy, on account of the cachexia, and the occurrence of tumours elsewhere. Where the primary disease is in the mediastinum or lung, or in the pleura itself, the diagnosis may be much more difficult, and the hæmorrhagic nature of the effusion may then be an important fact.

iii. *Simple.*—Theoretically, a simple inflammation, if of sufficient intensity, may cause hæmorrhagic effusion in the pleura, as in other places, but, as a matter of fact, it is rare.

In some of the instances in which it was apparently simple, the course of the case ultimately proved it to have been of tubercular nature.

iv. *Miscellaneous.*—All the other causes of hæmorrhagic effusion are very rare.

It has been found associated with general hæmorrhagic conditions; for instance, it might occur in the course of malignant (that is to say, hæmorrhagic) specific fevers, but it must be very rare. Trousseau describes it, and Wilson Fox gives a reference to some cases recorded by Blumenthal. Fagge removed a pint of dark brown fluid from a patient who had pleurisy as a sequel of scarlet fever, the patient making a good recovery. Many authorities do not even refer to it.

Of hæmorrhagic effusion in the course of purpura and hæmophilia, little is known, but in the course of severe scurvy several instances are recorded, though in these cases there is rather an actual hæmorrhage into the pleura than a hæmorrhagic effusion.

Bright's disease is given as an occasional cause, and a good instance of this is recorded by Poulin[1] in connection with granular kidney, and Lebert also refers to the subject. I do not know any instance of it in connection with any other form than that of chronic interstitial nephritis (granular kidney). Its occurrence is generally referred to the degeneration or disease of the vessels, and if this be so, it might also occur in cases of general atheroma; a case attributed to this cause is reported by Dugnet.[2]

In cirrhosis of the liver, it has been recorded, but there is reason to believe that in many of these instances the cause was really tubercle.

Where an effusion occurs in connection with infarcts, it is likely to be blood-stained; but, as a matter of fact, infarcts, though they frequently lead to dry pleurisy, rarely cause effusion.

Hæmatoma of the Pleura.—This condition is described by Netter,[3] and a case is recorded by Moutard-Martin,[4] but I have never seen an instance of it, and do not know of any specimen of the kind in our museums. It has been compared to pachymeningitis hæmorrhagica. If the condition exist, it must be one of extreme rarity.

70. HÆMOTHORAX.

Hæmothorax, hæmorrhage into the pleural cavity, must be distinguished from the condition in which the effusion is not blood, but merely blood-stained.

With but few exceptions the hæmorrhage is due to the lesion of some large vessel within the thorax.

The cases fall into two groups, according as they are traumatic or not.

1. *Traumatic.*—In these cases the vessel is wounded as the result of a fall, crush, bruise, or penetrating wound.

With a penetrating wound, as, for instance, a stab or bullet wound, any large vessel, or even the heart itself, may be injured; then the hæmorrhage is profuse and the result probably fatal in a very short space of time. If a smaller vessel be opened, for instance the intercostal or internal mammary, the hæmorrhage, though free, will not be so profuse, nor the symptoms so severe.

The lung is often wounded, but unless one of the large branches of the pulmonary vessels be also injured, the wound rarely leads to hæmorrhage into the pleura. When the lung is injured, as for instance by a stab, the blood effused into

[1] *Bull. d. l. Soc. Clin.*, 1879, p. 81.
[2] *Gaz. des Hôpit.*, 1885, Aug. 4.
[3] Charcot, *Méd.*, 1021.
[4] *L'Union médic.*, 1884, No. 50.

the vesicular structure of the lung clots at once and forms a solid mass, so that the opening into the pleura is sealed; and although free hæmorrhage may take place from the bronchial tubes, bleeding into the pleura rarely occurs to any extent.

A fall, a crush, or a blow may rupture a large vessel, and that too without much external mischief, and sometimes without any obvious damage at all.

Thus I have seen the aorta completely divided, all except an eighth of an inch of its coat, without any external bruise upon the body, in a man who had had a heavy fall; hæmorrhage then extended into the sheath of the vessel, and ran along the mediastinum and round the ribs behind the pleura, though it did not actually perforate the pleura. The man lived for some hours.

With fractured rib, hæmorrhage into the pleura is rarely copious, unless several ribs be broken. In most cases, though the lung may be seriously damaged and free hæmoptysis occur, and even though air escape from the lung and produce widespread subcutaneous emphysema, still little, if any, blood enters the pleura, nor, it may be added, does pneumothorax result.

These traumatic cases need not be referred to in further detail here, for they are fully treated of in works on surgery.

2. *Non-traumatic cases.*—In the non-traumatic cases the rupture of the vessel is caused by some previous intra-thoracic lesion, *e.g.*, the rupture of an aneurysm, the erosion of a vessel by a new-growth or abscess, the bursting of a varicose vein on the thoracic walls; in some rare cases it is the result of some general hæmorrhagic condition.

Aneurysms of any part of the thoracic aorta, or even of the large branches of it within the thorax, may rupture into the pleura, but, as a matter of fact, it is chiefly with aneurysm of the first part of the arch of the aorta that rupture into the pleura occurs, and that, of course, on the right side. Aneurysms of the middle part of the arch usually rupture into the trachea; those of the descending part of the arch, and rest of the thoracic aorta, into the left lung or bronchus.

Cancer also may open a vessel, but generally a communication is also formed with the œsophagus, trachea or bronchi, rather than with the pleura; and the same is true of abscesses or suppurating glands.

There are also rare cases recorded in which an abscess in connection with a carious rib has opened the intercostal artery and caused free hæmorrhage.

With a mediastinal tumour, which compresses the larger veins, a collateral circulation may be formed, and under these circumstances the internal veins of the thorax—for instance, the internal mammary, the intercostal, or azygos veins—may become greatly dilated or even varicose, and one of these varicosities may rupture.

Diseases of the lung very rarely lead to hæmorrhage into the pleura.

A curious case is recorded by Perry, in which, in extensive gangrene of the lung, a large vessel was opened and several pints of blood were found in the pleura. I have seen a similar thing occur in chronic phthisis from an aneurysm in a chronic cavity. This cavity had communicated already with the pleura and led to pyo-pneumothorax, for which the side had been opened. Very free hæmorrhage occurred on two or three occasions, and in the last caused the patient's death.

A very interesting, but also the rarest, group of cases is that in which hæmorrhage into the pleura occurs without gross lesions of large vessels, but by oozing, as it were, from numerous small ones in hæmorrhagic states. Thus it might be met with in the course of the hæmorrhagic specific fevers, scurvy, hæmophilia, and possibly purpura, as well as in various profound anæmias, of which leucocythæmia may be taken as the type.

In hæmorrhagic fevers, effusions occur, and they are sometimes blood-stained; but although hæmorrhage may take place from almost any free surface in the body, actual hæmorrhage into the pleura is, I believe, almost unknown.

The same is true, I believe, also of purpura, hæmophilia, and other forms of grave anæmia.

The best instance of this kind of hæmorrhage is met with in scurvy, in which very large effusions of blood may occur into the pleura, as into the pericardium, and, strange to say, in not a few instances recovery has taken place.

The signs of hæmothorax fall into two groups:

1. Those of severe hæmorrhage.
2. Those of a rapidly-forming effusion.

The signs of severe hæmorrhage are syncope and pallor, with an almost imperceptible pulse. If the hæmorrhage be very profuse, the patient will not rally, but will die in a few minutes.

This is probably to be attributed to the shock as much as to the hæmorrhage; and sudden death may occur from shock even when the hæmorrhage is not very severe.

As soon as the patient rallies, the symptoms of embarrassment of the respiration are added, viz., dyspnœa and cyanosis.

If life be prolonged, so that the patient rallies and the bleeding stops, the symptoms may subside and become simply those of a large and rapidly-formed effusion.

In many cases the access is very much like that of pneumothorax, the patient being seized with pain in the side and severe dyspnœa.

The severity of the symptoms depends upon the amount of blood effused and the rate of its effusion.

The Result of the Hæmorrhage.—The blood, shortly after effusion, coagulates and separates into serum and clot, the latter gravitating to the lowest parts of the cavity. The serum is readily absorbed, and so in some cases is the whole effusion. This has been experimentally shown in animals, and has been also observed in man, so that in the course of a few days a large hæmothorax may completely disappear. More commonly, however, although the serous part may be absorbed quickly, the clot takes a much longer time to disappear within the pleura, just as it does in the subcutaneous tissue or elsewhere in the body.

Except coagulation, and gradual disintegration in the process of absorption, the blood undergoes no changes of itself, nor does it excite inflammation, unless it be infected. But as blood offers a very suitable soil for the growth of pathogenic organisms, it is very likely to become infected with pyogenic bacteria; then acute inflammation will follow, and the symptoms of empyema develop. Of course infection is very likely to occur where there has been a penetrating wound, or where there is some disease, within the thorax, of a part communicating with the external air, as, for example, of the œsophagus, trachea, or bronchi. In the same way, great care is necessary if hæmothorax be tapped to see that the needle and instruments used are perfectly aseptic.

The diagnosis is easily made; for even if there be no injury, there is usually the history of some disease, like aneurysm or new-growth, to suggest the cause; while the sudden onset of the condition is unlike anything except pneumothorax, from which affection hæmothorax can be easily diagnosed by the physical signs.

In traumatic cases there is one condition which may present difficulty, viz., rupture of the diaphragm. In this case the injury will produce profound

shock, and in the next place the passage of the organs of the abdomen into the thorax may cause great dyspnœa and puzzling physical signs, which, however, are more likely to suggest pneumothorax than hæmothorax.

Thus I saw a case in which it was thought the patient had pneumothorax, because of the great displacement of organs and the amount of dyspnœa. The symptoms had come on after the patient had been run over; the physical signs were puzzling, for though the chest was resonant in parts, there were areas of dulness which did not at all correspond with a pneumothorax, nor did they agree either with an effusion of blood. It was impossible to do anything to give relief to the patient. After death it was found that the diaphragm on the left side had been extensively ruptured, and that the spleen, stomach, and parts of the intestines had passed into the pleural cavity.

The prognosis depends to a great extent upon the cause; but the presence of blood in the pleura does not *per se* necessitate a grave issue. If the hæmorrhage cease, the blood may be readily and completely absorbed. If inflammation occur, suppuration will probably take place; the case then be converted into one of empyema, and the prognosis will be much the same as in that affection.

Treatment.—The treatment will necessarily be largely modified by the cause. Surgical procedures are impracticable, for in most cases the bleeding vessel could not be found, or, if found, could not be ligatured. If the case be not immediately fatal the hæmorrhage will soon cease; and in the stopping of the hæmorrhage, the pressure of the blood already effused into the pleura plays an important part, so that it is well not to be in too great a hurry to remove the blood from the pleura, if it be possible to wait.

In such a case, while waiting, the patient should be kept absolutely at rest in bed, in such a position as is most comfortable to him. To relieve the excitement and distress, some opium should be given by the mouth, or an injection of morphia *sub cutem*, while the pulse should be watched and collapse warded off by the administration of a small quantity of stimulants from time to time.

Ice-bags have been recommended to be applied to the chest, but they are quite useless; nor, under ordinary circumstances, is the administration of ergot or ergotine of the slightest advantage. If the pressure symptoms be urgent, and something must be done to give relief, the fluid may be removed by paracentesis, and for this the aspirator will probably be necessary; but no more suction should be used than is just enough to make the fluid flow, nor should more be taken away than is sufficient to give some relief. Above all things, it is necessary to see that the needle and tubes are absolutely clean, so as to avoid all possibility of infection. If by this means relief can be given to the urgent symptoms, in a short time the hæmorrhage will cease, and is not then likely to recur.

The further treatment of hæmothorax will depend upon whether inflammation follows the hæmorrhage or not.

In the cases in which inflammation follows, it will be almost certainly of a suppurative character, and the case will become converted into an empyema, which will have to be treated on general lines. So far as the effusion alone is concerned the result will be much the same as in empyema, and many of these cases recover.

If the blood have not been infected, and no inflammation occur, the question will then arise whether the blood should be left alone to be absorbed, or whether its removal could be accelerated by any operative interference.

Considering that in many cases blood is very readily absorbed from a healthy pleura, it is as well to wait for a time and see what is going to happen. In the cases in which absorption is slow, paracentesis may be performed, and the removal of the fluid thus accelerated.

The only other course to pursue is to open the side, wash out the blood clots, and deal with it in the usual way afterwards.

Most of the cases treated in this way are those which follow accidents. It will then be necessary to consider how far the extensive injury and bruising to the thoracic walls would militate against an effectual operation. However, even in very severe cases, successful results have been attained by modern surgery.

My colleague, Mr. Howard Marsh, had a very serious case, in which several ribs were fractured and the pleura filled with blood ; the side was freely incised, the blood clot removed and the side emptied. Extensive as the operation was, the result was in every way successful, and recovery was very rapid.

71. CHYLOTHORAX.

In chylothorax the fluid has a milky appearance and contains much fatty matter. It is alkaline in reaction, has a specific gravity of about 1015 to 1018, and is of a pale yellowish colour. The milky appearance is due to the suspension in the fluid of numerous fat-globules, and occasionally of fatty crystals (cholesterin). On standing, it frequently coagulates spontaneously, and upon the surface there forms a layer of varying thickness like thin cream. The actual percentage of fat which the fluid contains varies much. In some cases the fluid is actually chyle, and contains the same percentage of fatty matter. In others the fluid has more the character of a milky serum, and the percentage of fat is less.

There are two conditions which require to be distinguished, though very often they are described by the same term.

In the one the fluid is simply chyle, and the effusion is due to the leaking of chyle into the pleural cavity, owing to the rupture of the thoracic or one of the larger lymphatic ducts ; this has been called "*Hydrops Pleuræ Chylosus.*"

In the other the fluid is rather a milky serum, in which the fat is apparently due to the fatty degeneration of cells. This is called *Hydrops Pleuræ Adiposus* (Quincké).[1]

1. Hydrops Chylosus.—This is a rare condition. Bargefuhr,[2] writing in 1895, could only find 11 cases of true chylothorax recorded.

In these cases there is a direct communication of the thoracic duct, or one of the larger lymphatic ducts, with the pleural cavity, so that the fluid present is actually chyle, as is shown by its composition. The following is an analysis of such a fluid made by Yvon.[3]

In 1000 parts.

Water,			913
Salts,			7·1
Organic Matter—			
Coagulated albumen,	68·0		
Cholesterin,	3·8	7·2	76·7
Fatty substances,	3·4		
Nitrogenous substances,	1·5		
Loss,			2·58

[1] *D. Arch. f. kl. Med.*, xvi.
[2] *Virch. Arch.*, 1895, vol. liv. p. 410.
[3] *Soc. méd. des Hôpit.*, 1887.

In the majority of cases the rupture is due to injury, but in a few to disease.

The following case, the result of injury, is recorded by Thaden :[1]

A man, aged 31, was run over and had many fractured ribs. Pneumothorax was produced, and fluid developed on the right side.

On the first paracentesis, a short time after the accident, about 85 ounces of almost pure blood were removed. On the second paracentesis, a few days later, nearly 7 pints of clear, blood-stained fluid were removed, containing numerous small fatty bodies floating in it. On the third paracentesis, a little later, when 115 ounces were removed, the fluid was nearly clear.

The patient died, and on *post-mortem* examination a large effusion was found, filling the whole pleura. From the top of this to about the level of the third costal cartilage, a mass was found floating in the fluid as large as the fist, and resembling cream or clotted milk. This, when removed, floated in the water. The thoracic duct could not be found. Analysis of the fluid showed that it contained 3·71 in the 1000 parts of fat, and 0·43 of grape sugar.

In many cases the injury is not so grave as this, and it seems probable that the thoracic duct may be ruptured without much external sign of serious damage at all.

Erosion of the thoracic or large lymphatic ducts may be brought about by disease. Neuenkirchen [2] records a remarkable case, probably of this nature.

A woman of 47 had removed from her chest, in the year 1884, 35 ounces of milky fluid. In the year 1889 she came under observation again, when 70 ounces of yellowish milky fluid were again removed. It contained numerous fatty granules, but no fatty cells, was alkaline, had a specific gravity of 1016, on standing coagulated spontaneously, and formed a creamy layer. It contained 3 per 1000 of a butter-like fat.

Two other paracenteses, yielding smaller amounts of fluid of the same character, were performed in the course of the next two months. She then left the hospital, and though the chest contained fluid, was not tapped again for nearly four months, and then 50 ounces of similar fluid were taken away. Twice during the ensuing year she was tapped, with similar results. Then it was thought desirable to repeat paracentesis no more, as no improvement seemed to have been produced. The subsequent history of the case is not recorded ; but at any rate this patient must have had chylous fluid in the thorax for a period of about six years at least, and seemed to suffer no discomfort or damage to health, except that when the fluid reached larger dimensions than usual it caused some shortness of breath, and required paracentesis.

In some of these cases the thoracic duct is not itself in fault, but the rupture is probably in one of the large lymphatics, elsewhere in the thorax.

Thus in a case of Sidney Martin's,[3] in which there was chylous ascites as well as chylothorax, the thoracic duct was not ruptured, but the left jugular and sub-clavian veins were completely obliterated, and a clot extended for some little way down the thoracic duct. The fluid, it was believed, must have gained access to the pleura from some of the radicles of the thoracic duct, though the communication could not be discovered. A similar case is recorded by Turney,[4] in a case of cancer, in which the thoracic duct was greatly dilated, but not ruptured, and was plugged at the entrance into the vein.

It is probable that tubercle or cancer of the parts near the duct may cause its erosion, and certainly chylothorax is often found associated with these diseases, but I do not know of any instance in which the erosion of the thoracic duct has been actually demonstrated.

A unique case is recorded by Erb,[5] associated with lymphangiectasis of the leg, in which all the lymphatics were found dilated along the abdomen to the receptaculum chyli, and upwards to the thoracic duct, the lymphatics of the mediastinum and root of the lung being greatly dilated as well as those of the pleura, some of which opened directly into the pleura.

[1] *Arch. f. kl. Med.*, xix.
[2] *Petersburg Wochensch.*, 1890, No. 51.
[3] *Path. Soc. Trans.*, xlii. 93.
[4] *Path. Soc. Trans.*
[5] Quoted by Bargefuhr, *loc. cit.*

2. Hydrops Adiposus.—In these cases the fluid contains less fat, and is really a serous fluid mixed with a certain amount of fat. The fat is found in the form of a granular detritus, forming a sort of emulsion in the fluid, and there are besides a number of cells in a condition of advanced fatty degeneration.

Some of these cases have been associated with cancer.

Thus in Bögehold's[1] case, a man of the age of 43, a yellowish fluid was removed from the pleura, which formed on standing a layer of fat a millimetre thick. On dying, this patient was found to have cancer of the stomach, with secondary cancer of the pleura. The thoracic duct was intact, and the fat in the fluid was attributed to the degeneration of the cancer cells, numbers of which were found in advanced fatty degeneration in the fluid.

In other cases there is no new-growth to explain the condition, and the cells are assumed to be derived from the pleura itself.

Thus Debove[2] records a case in which 28 ounces of fatty fluid were removed, containing fat drops, fatty detritus, and some cholesterin crystals. On the *post-mortem* examination the pleura contained 80 ounces of the same fluid; the surface of the pleura was covered with exuberant fatty granulations, and the fluid contained a number of these cells in a condition of extreme fatty degeneration. The case was described as one of fatty pleuritis.

Cases of this kind may be compared with those which have been already described, in which, in the later stages of a long-standing inflammatory effusion, the fluid is found to contain cholesterin.

Treatment.—No special treatment is required. They have to be dealt with on the lines of ordinary pleuritic effusion.

The result depends greatly upon the cause; but where cancer and tubercle are absent, and where we have not to deal with results of severe injury, the case may run a favourable course. Thus it may happen that one or two paracenteses may lead to cure; on the other hand, repeated paracenteses sometimes seem to have no effect at all, the fluid returning as soon as it is removed, and, as in one of the cases referred to, the effusion may exist for several years, and retain all the time its peculiar character.

72. PNEUMOTHORAX.

Pneumothorax is a general term used to describe the condition in which the pleural cavity contains air. If fluid be present also, the prefix "hydro," or "pyo," is added, according as the fluid is serous or purulent respectively, the disease being then called hydro- or pyo-pneumothorax.

The cases in which there is a free communication through the chest walls between the external air and the pleura—the result of operation, as in the opening of an empyema, of accidental injury, or of the rupture of an abscess in the chest walls—are not, as a rule, included under the term pneumothorax, which by common consent is reserved for those cases in which the source of the air is not so obvious.

History.—The name "pneumothorax" was invented by Itard in 1803, though the condition was known to many earlier writers. By one and all, the existence of fluid was regarded as essential, and the presence of the gas attributed to exhalation from the fluid. It is to Laennec that we owe the first accurate study of the condition, and there is not much to add to his original description of it. Subsequent writers have concerned themselves chiefly with explanations of the various physical signs observed. Of recent years clinical observation has shown that the disease is not so rare as was once thought, and that it may develop without the striking symptoms with which it was formerly supposed to be necessarily associated.

[1] *Berl. klin. Woch.*, 1878, No. 24.

[2] *Gaz. des Hôp.*, 1881, No. 49.

ETIOLOGY.—The theory that the air in the pleura is derived by exhalation from the fluid in the chest has long been recognised as inaccurate. It can indeed explain only one class of case, probably the rarest of all, viz., that in which gangrenous decomposition occurs in a purulent fluid.

When air is found in the pleura, it must, therefore, have gained access to it from some air-containing organ within the body.

These organs are, of course, chiefly the lungs and air-tubes; but under certain conditions the air may be derived from the œsophagus, or even from the stomach or intestines.

The lungs themselves may be ruptured, either as the result of an injury or of disease; in the former case, a stab, broken rib, or crush may be the obvious explanation; in the latter, the rupture of a cavity lying near the surface of the lung is the commonest cause. Besides these, it is possible for the pleura to be ruptured by violent expiratory efforts; for instance, after violent coughing as in children with whooping-cough, or after tracheotomy.

Phthisis.—In the overwhelming majority of cases, pneumothorax is the result of the rupture of a tubercular cavity lying close beneath the pleura.

Saussier's statistics, so often quoted, attributed pneumothorax to phthisis in 62 per cent. of all cases. But of Saussier's[1] 131 cases, no less than 29 were referred to empyema; and as pneumothorax after empyema is so uncommon, except after incision of the side, we may fairly deduct probably the whole of these cases from his numbers, which would then yield a percentage of 81. Behier's figures (50 out of 58), and also Biach's (715 out of 918), yield a percentage of 80.

We shall, I think, be near the truth if we accept 90 as the percentage of cases of pneumothorax which may be fairly referred to phthisis. This is the percentage my own statistics yield, and it is in accord with those of Walsh.[2]

If, from the remaining 10 per cent., we deduct, on the one hand, all the cases in which the pneumothorax is the result of gangrene, or acute inflammatory destruction of the lung not tubercular in nature; and, on the other hand, those in which it is the consequence of injury to the lung or air-tubes, the relative rarity of the other assigned causes becomes evident, 2 or 3 per cent. must include them all; while pneumothorax resulting from violent respiratory efforts, *e.g.*, after tracheotomy, or in the course of whooping-cough, though somewhat more frequent, is still very rare.

The following figures show the relative frequency of these causes *inter se*: of Saussier's 102 cases (*i.e.*, 151 cases – 29 cases of empyema) the cause was phthisis in 81; of the remainder, gangrene in 7, emphysema in 5, infarct in 3, hydatid, abscess, cancer, hæmothorax, in 1 each; lung-hepatic fistula in 2.

For practical purposes it is with pneumothorax as it occurs in phthisis that we have especially to deal, and it is upon this that the general account given of the disease will be based; but before passing on to this it will be well to consider the other causes of pneumothorax, for they are of theoretical importance as well as of practical interest.

Empyema.—Where air gains access to the pleura, either by an empyema bursting externally or after incision, the cases are not usually regarded as instances of pneumothorax, and they have been already considered under "empyema"; when an empyema bursts through the lung, a communication is made by which air may pass into the pleura, but, as a matter of fact, this occurs by no means as frequently as might be expected.

[1] *Cf.* Wilson Fox, note, p. 1104.

[2] West, *Lancet*, May 3, 1884.

The reasons of this have been already considered. They are (1) the defective movement of the side; (2) the position of the perforation, which is usually in the lower part of the lung; (3) the greater readiness with which the air enters the upper part of the lung rather than the lower; and (4) the presence of what may be described as a fluid-valve, for the pus enters some little distance into the air-tubes, and would have to be forced back before the air could enter the pleura. Satisfactory statistics are not easy to obtain.

The frequency, as stated by Biach, is $\frac{45}{918}=5$ per cent.

Saussier's percentage is higher than this, nearly 20 per cent., but this is out of all proportion to the experience of the present day.

Destructive Diseases of the Lung other than Phthisis.—Of these, gangrene is the chief, and abscess the next. Embolism, infarct, croupous-pneumonia, or broncho-pneumonia, can only produce pneumothorax if they are associated with suppuration, that is to say, abscess. Cancer or hydatid might, in the same way, lead to a communication between a bronchus and the pleura.

Gangrene.—This is very often deep-seated and far away from the pleura, but if it should be near the surface, the pleura will become involved and perforation take place; the resulting inflammation will then be of a septic character, and the result invariably fatal.

Abscess.—This, also, is usually deep-seated, and when near the surface it often does not spread with any great rapidity, so that the pleura becomes inflamed and adherent over it, and when the abscess bursts, it bursts into a localised portion of the pleura, which is shut off from the general cavity. In the same way it not rarely happens that the abscess does not at first form a free communication, or any communication, with the bronchus, so that when the pleura is opened the pus may escape into the pleura, but no air with it. In any case in which an abscess forms, it is much more likely to discharge itself through the air-tubes than to open into the pleura.

Pneumonia.—Croupous-pneumonia is not of itself a destructive inflammation of the lung, and, therefore, cannot lead to pneumothorax. In the cases in which abscess and gangrene occur, perforation of the pleura may happen; but these are extremely rare, and in the majority the patients die before any change in the pleura is effected. I have seen one case myself in which the pleura had become involved in the gangrenous process, and was riddled with perforations. Under these circumstances the resulting inflammation of the pleura is septic, and the patients invariably die.

Broncho-pneumonia.—In the same way this very rarely leads to destructive inflammation, and when it does, the broncho-pneumonia is probably itself caused by septic organisms. A case of this kind has been described by Steffen in the course of measles.

Barthez and Rilliet say that broncho-pneumonia is the commonest cause of pneumothorax in children. For this there may be two possible explanations—one that which has been already given, viz., that the broncho-pneumonia is of a septic and destructive character, and the other a different one, viz., that the broncho-pneumonia and the rupture of the lung are themselves both independent results of some other affection, as, for example, whooping-cough.

Pneumothorax after typhoid fever is connected with small broncho-pneumonic patches, and these are probably of septic origin.

Cancer.—This usually invades and destroys the lung, and thus shuts off any communication with the bronchial tubes, and, when it spreads to the pleura, infiltrates it in the same way. It follows, therefore, that it must be excessively rare for cancer in the lung to produce pneumothorax, for it must on the one hand lay the pleura open, and on the other communicate with a bronchus.[1]

Hydatid.—Hydatids, in the same way, rarely lead to pneumothorax, for though they may discharge themselves into the pleura on the one side, or into the air-tubes on the other, they but rarely discharge themselves in both directions at once, and even when they do, the communication is rarely of such a kind that air can pass back into the pleura.

Instances, however, of this are given by Behier.[2]

Common Emphysema.—To the rupture of an emphysema bladder a considerable number of those cases of pneumothorax are referred, which occur in patients who do not present any evidence of disease of the lung; yet when we come to investigate these cases, it appears that the conclusion is rather a matter of inference than of evidence.

Experiment shows that though it is easy enough to rupture an emphysema bladder in a lung removed from the body, it is by no means so easy when the lungs are *in situ*, because of the support which the lung obtains from the chest wall and the parts about it.

[1] *Cf.* Wintrich, *Virch. Handb.*

[2] *Conférences.*

Post-mortem evidence also shows that even in lungs which have been supposed to be healthy, tubercular lesions, sometimes chronic and sometimes of recent date, may be found, to which the pneumothorax is due, and which no physical examination could have diagnosed during life. On the other hand, pneumothorax occurring as a complication in the course of a case of ordinary emphysema is almost unknown.

Biach [1] states that out of 2710 cases it occurred only once.

Zahn [2] has recently gone into this question, and can only find 4 cases recorded which seem to bear this interpretation, and of these only 2 appear to be really conclusive, viz., 1 recorded by Dittrich in 1854, and another by Fraentzel in 1877.

We may thus conclude that ordinary emphysema as the cause of pneumothorax may be practically disregarded, and that, when during life this seems to be a possible interpretation, it is more likely that the real cause is to be found in some other affection of the lung.

Violent Respiratory Efforts.—Of course, where the lung is diseased, violent respiratory efforts may determine the actual rupture into the pleura, and thus cause pneumothorax, but it does not necessarily follow that they can do so if the lungs be sound.

The question is thus raised whether it is possible to rupture a healthy lung by any force which respiration can bring to bear upon it. To this general question an affirmative answer must be given. The violent paroxysms of whooping-cough and the straining of parturition afford clinical evidence that the lung may be ruptured by expiratory efforts. The condition, however, which is commonly produced is not that of pneumothorax, but that of subcutaneous emphysema.

A careful experimental study of this subject was made by Dr. Champneys [3] upon the lungs of infants. When the lung is over-distended and gives way, the air first makes its way beneath the pulmonary pleura, stripping it easily off for some distance from the surface of the lung. It then passes along the root of the lung to the mediastinum, and following thence the course of the cervical fascia, it reaches the subcutaneous tissue of the neck, whence it may spread over the whole body. If the pleura give way, the place of rupture is to be found usually near the root of the lung; but, although pneumothorax does now and then arise in this way, the usual result is that already described, namely, emphysema of the mediastinal tissue and of the neck. Sometimes by artificial respiration, after tracheotomy, the air takes the reverse direction, and tracking downwards from the tracheotomy incision along the deep cervical tissue, reaches the mediastinum, or even the subpleural tissue, and then the pleura may be ruptured and pneumothorax produced.

These experimental observations are confirmed by clinical experience. Dr. Champneys [4] records 27 necropsies after tracheotomy. Mediastinal emphysema was found in 3, and in 2 of these pneumothorax was present. The pneumothorax in one of them was double, and the cause of death. In 82 necropsies at the Children's Hospital mediastinal emphysema was found in 5, but pneumothorax in none. Wilks and Moxon mention 2 cases of pneumothorax after tracheotomy, and connect it with mediastinal emphysema. Dr. Angel Money, examining 28 similar cases with especial reference to this point, found mediastinal emphysema in no less than 16, and pneumothorax in 2, but in both of the latter the pneumothorax was associated with mediastinal emphysema. In most of these cases artificial respiration had been performed.

It seemed desirable, considering the delicate texture of the infantile lung, to repeat these observations upon the adult, and I did this with the result of confirming in the main the conclusions at which Dr. Champneys had previously arrived. I noticed also in my experiments the same important and curious fact to which Dr. Champneys had referred, viz., the difficulty of obtaining bursting pressure by means of water, owing to the permeability of the lung to water

[1] *Loc. cit.* [2] *Virch. Arch.*, 1891. *Cf.* also Gaillard, *Arch. gén. de méd.*, 1880.
[3] *Med. Chir. Trans.*, vol. lxv. [4] *Cf.* Bradshawe Lect., *Lancet*, August 1887.

while it was still air-tight. The lung seemed almost like a sponge, so that the water flowed from it with such readiness, when distended, that the pressure could not easily be maintained.

The pressures at which rupture took place varied much in different cases. In four cases it reached $2\frac{1}{2}$ inches of mercury, in one 2, and in another 4 ; so that what Dr. Champneys found in infants is true also in adults—that the normal resistance of the lung is widely different in different persons. My own observations were made upon the lungs after removal from the body, and the bursting pressures thus obtained are, no doubt, much below those which would be required when the lungs are *in situ*. In the only case in which I was able to attempt this experiment upon the dead body, the air did not appear in the neck until a pressure of nearly 8 inches of mercury was registered, and pneumothorax was subsequently found on both sides. This experiment agrees with the statement made by Hutchinson, that the cells of the lung can resist a pressure of from 3 to 9 inches of mercury when expanded as in health. A pressure of 8 or 9 inches of mercury is far beyond anything that even an adult can produce by expiratory effort ; and it is clear, therefore, that it can only be in cases where the normal resistance of the lung is greatly reduced that, in the absence of any disease of the lung, pneumothorax can be produced.

The lung is therefore capable of resisting in most cases the highest pressure that the most violent expiratory efforts can bring to bear upon it, and the question may well arise whether, in those cases in which the lung has given way, the absence of a local lesion can be without hesitation assumed. However this may be, it is clear that when rupture of the lung occurs, the result is usually sub-pleural, mediastinal, and cervical emphysema, and only very rarely pneumothorax. This conclusion clinical observation abundantly confirms. Although every text-book speaks of emphysema among the complications of whooping-cough, pneumothorax is, by most writers, not even mentioned. So far as parturition is concerned, Dr. Matthews Duncan told me that, although emphysema is by no means uncommon, he had never yet seen a single case of pneumothorax produced during parturition, and this experience has been confirmed by that of other obstetricians.

I append the actual notes made at the time of experiment :—

1. Left lung ; adult man about 50. Death from septicæmia after osteomyelitis of femur. Lower lobe a good deal collapsed, especially on posterior and lower part ; no consolidation, no old adhesions. Lung expanded readily in all parts except where there was collapse. The pressure was raised to $2\frac{1}{2}$ inches of mercury, and the lung then burst at the posterior and middle part of upper lobe about 2 inches from root of lung. When rupture occurred the pleura was suddenly stripped off over a space about the size of a five-shilling piece, and then it gave way, the hole measuring about an eighth of an inch.

The tube was next tied into the bronchus of the lower lobe. As the pressure rose some of the collapsed portions expanded, but not all. The lung finally ruptured suddenly on the posterior border, where the lung lies at the side of the spinal column. Here the pleura was suddenly stripped off the lung for the whole length of the lobe (6 inches), and this gave way with a rent about half an inch long. On opening the raised part of the pleura the lung was found to be ruptured in several places : for the most part the holes were minute, but in one, which corresponded with a small portion of collapse, the hole was nearly an eighth of an inch in diameter.

2. Left lung ; adult man about 50. Lung a good deal collapsed ; small fibrous nodules felt through the pleura, scattered throughout the lung ; an old fibroid cicatrix at the apex. The lung distended readily (even the collapsed portions) and resisted a pressure up to 4 inches of mercury. At this pressure it gave way at the posterior and middle portion of the upper lobe ; the pleura stripped off for a space about the size of a shilling, and then burst by a small aperture. The tube was fixed in the lower lobe, and the pressure was now raised to nearly 5 inches of mercury before any considerable rupture occurred. This took place at the posterior border, close to the root of the lung, but the pleura did not strip off, as in the last case. Before this pressure was reached many small patches of interstitial emphysema developed all over the surface of the lobe, but none of them burst. On section a fibrous nodule was found in the apex, and small nodules (so-called "peri-bronchitis") were scattered throughout both lobes.

3. Right lung of a boy. The cannula was tied in the bronchus and the lung inflated ; at a pressure of $2\frac{1}{2}$ inches of mercury it burst. The air came out of minute apertures around the inferior bronchial vein and along the posterior and mediastinal borders ; some air seemed to come from the vein itself.

The inferior lobe burst first, but there was a large slit accidentally made in the upper lobe before the experiments began. Where the lung burst, except close to the root, the pleura was first raised. It was not stripped off in large pieces, but in pieces varying in size from a large pea downwards.

4. Left lung of male, aged about 55. Died after gastrostomy for stricture of œsophagus. Upper lobe particularly emphysematous, lower lobe not so much so; otherwise healthy. Air was forced into the lower lobe only, as the upper one had been cut. At a pressure of 2 inches of mercury the pleura was suddenly slightly raised in two or three places near the vein, and air came up also through the vessels. The holes were very minute in the lung-substance, and the pleura raised was about the size of a pea in each place.

5. Left lung of male, aged 66, who died after cancer; emphysematous, but otherwise healthy. At a pressure of 2½ inches of mercury the pleura was suddenly raised up over an oval space, 2 inches in length and three-quarters of an inch in breadth between the upper and lower lobes near the root of the lung.

6. Left lung of male, aged about 50, who died suddenly from the bursting of an aneurysm of the aorta situated where the thoracic passes into the abdominal aorta. The aneurysm burst into the right pleura. Air was forced in, and at a pressure of 2½ inches a part of the pleura 1½ inches long and 1 inch wide was suddenly raised from the lower lobe close to the root. At the same time air came up through both artery and vein. The hole in the lung itself could not be seen; the hole in the pleura, which was raised, was a quarter of an inch in diameter.

Injury.—Any injury which lays open the pleura, either through the chest walls or by laceration of the lung, with or without an external wound, may possibly lead to pneumothorax, but there are many exceptions, so that pneumothorax need not necessarily follow.

As these exceptions are of great theoretical importance, I will refer to them here.

Wounds of the chest walls fall into two groups, according as they are punctured or open.

1. *Punctured wounds* such as result from a stab are often associated with subcutaneous emphysema, which may extend over a considerable portion of the chest and even spread widely over the body. They may be also associated with a certain amount of hæmoptysis. Yet pneumothorax, as the result of a stab, is by no means common.

It is often assumed that when surgical emphysema results from a stab, pneumothorax must be a necessary antecedent. Yet this assumption is not based upon facts; for the symptoms of pneumothorax are lacking, there is no displacement of organs, and *post-mortem* examination shows that pneumothorax is absent.

To explain these difficulties, it is assumed either that air has been sucked in through the external wound into the subcutaneous tissue or that the pleural cavity has been obliterated by adhesions. In respect of the latter theory we have no right to assume the presence of adhesions in persons who have previously been perfectly healthy, and *post-mortem* examination often shows them to be completely absent. In respect of the former theory, although wounds in the axilla may, it is true, in consequence of the sucking in of air by movements of the arms or of the chest-muscles during respiration, lead to surgical emphysema in the immediate neighbourhood, yet anything of this kind in the lower part of the chest is practically unknown, and the theory rests upon pure supposition.

The subject has, moreover, been submitted to experiment. Biermer [1] stated many years ago that it was only some punctured wounds of the chest walls and lungs that caused pneumothorax.

Wintrich [2] showed by experiments that if the lung were injured by a sharp, not too large or broad-pointed instrument, no pneumothorax resulted, although surgical emphysema might follow.

The same experiments were repeated with the same result by Fraser.[3]

We must conclude, therefore, that pneumothorax after punctured wounds of the thorax and lungs is the exception rather than the rule, as Biermer maintained many years ago.

[1] *Ztsft. f. Heilk.*, 1869, ii. 111.
[2] *Loc. cit.*, p. 337.
[3] *Penetrating Wounds of Chest*, London, 1859.

2. *Open wounds* of the chest also, sometimes even of considerable size, may lay the pleura open, and yet pneumothorax may not follow. It is true that in most cases pneumothorax occurs, but in some it does not. Experimental investigation has not led to altogether concordant results, though the majority of investigators state that the lungs collapse, and pneumothorax results immediately that the incision is made into the pleura. Other observers,[1] however, state that incisions of some considerable extent may be made without pneumothorax necessarily following.

In most of these cases the lung is, of course, injured as well, and there are some very remarkable instances in which, in spite of extensive injury both to the lung and to the chest walls, no pneumothorax occurred. A very striking instance of this kind is recorded in St. Bartholomew's Hospital Reports for 1876.

A wood-turner, aged 33, stabbed himself in the head, chest, and abdomen with a chisel. The wounds of the head and the abdomen were not very severe, but that in the chest, situated on the left side, between the fifth and sixth ribs, was 3 inches long. Through it the lung could be easily seen, wounded and bleeding. The wound in the chest was closed with strapping and oil lint; two days later there was subcutaneous emphysema, but this soon cleared up, and in four weeks the patient recovered completely without any further trouble.

It is remarkable in gun-shot wounds in the chest that pneumothorax is not infrequently absent. In the history of the War of the Rebellion,[2] out of 11,549 cases of chest wounds, pneumothorax only gave trouble in about half a dozen cases. More frequently, instead of pneumothorax, hernia of the lung took place.

From the reports which have arrived from the Cape, the experience in the recent war appears to be the same. Bullets have completely traversed the chest, wounding the lung both on entrance and exit, yet without producing the grave symptoms that might have been expected, provided the big vessels escaped injury, and recovery has been rapid and complete.

A medical friend sent me the following deer-stalking experience. A stag was shot, but ran for two hours before it dropped, though the bullet was found to have passed completely through the body from side to side, piercing both lungs.

Even as far back as 1809, P. J. Roux proved by experiment that a penetrating wound is not necessarily followed by pneumothorax. The following quotation[3] is interesting in this connection:—

"One may see the lung moving freely in respiration in an animal from which a great part of the wall of the chest has been removed. I have often on dogs made penetrating wounds on both sides of the chest larger than the opening of the glottis, and I know that an animal under these conditions lives a long time, and dies only of a sort of gradual asphyxia.

Injury to the Lung without external wound.—This is usually caused indirectly as the result of a fracture of the rib, but it may be produced directly by the original injury.

1. *Fractured rib.*—As with punctured wound, so with fractured rib, surgical emphysema is common and pneumothorax distinctly rare. In this case, however, there can be no doubt that the air must come from the lung itself.

Statistics are hardly necessary to prove this fact, but Turner[4] gives a series which may be quoted here:—

"Of 237 cases of fractured ribs, in 25 surgical emphysema developed, and in 28 there was hæmoptysis; in 4 only did pneumothorax occur, and these were all fatal."

Of course in many of these cases the wound into the lung is quite small, but it need not be, and in one remarkable case the laceration of the lung was most extensive, and yet no pneumothorax occurred.

Some years ago I made the *post-mortem* examination upon the body of a woman who had been run over by a wagon. There was no external wound beyond bruising, several ribs were broken, and a considerable amount of subcutaneous emphysema developed. The lungs had evidently been severely injured, for the patient had expectorated a great deal of blood. At the autopsy several ribs were found fractured, and the lung extensively torn over an irregular area about 2 inches in diameter; but in spite of this, the lung was everywhere in apposition with the chest walls, and there was a complete absence of pneumothorax. The pleural cavity was free from adhesions.

[1] Wintrich, p. 337. Biermer, *Ztsch. f. Heilk.*, ii. 111.
[2] Part I., Surgical Vol., p. 623.
[3] *Cf.* Paget, *Surgery of Chest*, 1896.
[4] *Lancet*, March 9, 1889.

2. *Laceration of the lung without a fractured rib.*—Even in these cases pneumothorax may be absent, and yet that the pleura is ruptured is shown by the occurrence of an effusion of blood into it, which has not been derived from the intercostal arteries.

The cases in which pneumothorax occurs from this cause, without any signs of external injury, are always interesting. Traube[1] has recorded one after a fall, and Paget refers to one or two other instances.

The damage done internally by a fall or a crush, without any external injury being visible, is very remarkable. I have elsewhere quoted a case in which, without any sign of bruising, the aorta was completely divided except for about the eighth of an inch ; and there are two very remarkable cases published of pneumothorax produced by the complete rupture of the bronchus, so that the lung was really torn off, as it were, from its root. In the two cases referred to, it is interesting to see how long life was maintained. In the first case the left lung was torn from its bronchus, and double pneumothorax resulted, yet the man lived for twenty-two hours, and in the other case there was pneumothorax on one side only, and the man lived for forty-one hours. The probable explanation[2] of the duration of life in these cases is that the air passed freely in and out through the ruptured bronchus.

Affections of Viscera other than the Respiratory Organs.—Where the air in the pleura is derived from some other source than the respiratory organs, the cases are rather curious than important.

Thus the œsophagus may open into the pleura—in some cases as the result of malignant disease ; in other cases as the result of suppuration in connection with foreign bodies ; and in some rare cases as the result of injury, or of rupture after vomiting.[3]

Where the source of the air is some viscus below the abdomen, it is usually after suppuration has occurred that the diaphragm becomes involved in the process and the pleura opened ; but even then, as a rule, the pleural cavity is shut off by the adhesions which form, so that when rupture takes place, it is into the lung rather than into the pleura, and pneumothorax does not occur.

Subphrenic pyo-pneumothorax is really a sub-diaphragmatic air-containing abscess, and not pneumothorax at all ; but if, as sometimes happens in these cases, rupture take place through the diaphragm, then, of course, a true pneumothorax will result.

Cases of this kind are described in association with ulceration in the stomach, duodenum, or intestines, whether of a simple or malignant character. They are also described in connection with certain hepatic abscesses, which have opened into the intestines on one side and the pleura on the other.

These cases require no further consideration.

THE MECHANISM OF PNEUMOTHORAX.—The mechanism of a pneumothorax is thus described :

"The elastic pulmonary tissue is always, to a certain extent, on the stretch ; it is always, so to speak, striving to pull asunder the pulmonary from the parietal pleura, but this it cannot do, because the air can have no access to the pleural cavities. When, however, the chest ceases to be air-tight, and air is introduced into the pleural chambers, the elasticity of the lung pulls the pulmonary away from the parietal pleura, and the lungs collapse."

This quotation expresses concisely the physiological doctrine of the day. It is not, however, completely correct, for the chest may cease to be air-tight and yet air may not enter the pleural cavity. If, however, air does penetrate between the layers of the pleura, the lungs collapse, as they are stated to do, by virtue of their own elasticity. Experiment upon animals, as well as observation of disease and injury in man, has established the truth of this latter statement. Still there are not a few instances in which, in spite of the pleura being opened, collapse of the lung does not occur, and air does not, therefore, enter the pleural chamber. In considering the whole question, these instances must not be disregarded. There are two great classes of such cases : the first, that in which the pleural

[1] *Ges. Beitr.*, 351, 900. [2] Biermer, Schweizer, *Ztsch. f. Heilk.*, ii.

[3] Two cases referred to in debate on Pneumothorax at the Medical Society of London, by Turner, *Lancet*, January 16, 1897.

cavity has been laid open by an external wound; the second, that in which, without any external wound, a communication between the lung or some other air-containing viscus and the pleura has been made by an internal lesion.

Instances of both groups of cases have already been given, and it is unnecessary to refer to them further.

That the pleural cavity should be opened, and yet that pneumothorax should not occur, is a great difficulty, and various solutions have been suggested.

It has been asserted that in such cases the pleural cavity had either been completely obliterated by previous disease, or that adhesions had rapidly formed round the wound, and thus sealed the pleural cavity.

These theories are *a priori* improbable; they have no clinical facts to support them, and they are disproved by *post-mortem* examination.

In the case of fractured ribs, in which surgical emphysema has occurred without pneumothorax, it has been also suggested that air does really enter the pleura, but that it is rapidly absorbed again, so that its absence after death is accounted for. When so careful a clinical observer as the late Dr. Hilton Fagge endorses this view, the need of a reconsideration of the whole question is evident. These explanations are unsupported by clinical observation, and are one and all disproved by *post-mortem* examination.

The fact, therefore, that the pleural cavity may be laid open, and yet that collapse of the lung with consequent pneumothorax may not occur, must be accepted, and an explanation for it sought.

The normal elasticity of the lung was calculated by Donders to be equivalent to 7 millimetres of mercury. Michael Foster rates it somewhat lower, viz., at 5 millimetres. Hutchinson agreed with the higher estimate as the result of direct determination upon the lungs of two criminals immediately after death. If, for the present purpose, the higher estimate be accepted, it follows that when the lung does not contract after the pleura is opened, it must be kept on the stretch by some force greater than 7 millimetres of mercury; and there seems to be no place for such a force to exist unless it be in the pleura itself; and if there, it is probably to be found in the cohesion between the two serous surfaces.

A theory of this kind seemed to admit of easy investigation, and it was put to the test in the following manner:—Two discs made of mahogany were taken.[1] They measured 2 inches (5 centimetres) in diameter, were smooth on the surface, and had a groove cut round the margin. Over these pieces of stomach were tightly stretched with the peritoneal surfaces outwards, care being taken to select pieces which would lie as smooth and even as possible. These two discs so covered moved very freely over one another; but when they were pulled straight asunder considerable force was required to separate them. In order to estimate this force, one of the discs was connected with the arm of a balance, and while the other was fixed fast, weights were placed in the scale-pan until the two were pulled apart. I found in some of my experiments that about 325 grammes were required. It was thought that the force which kept these discs together might have been atmospheric pressure, and in order to eliminate this, two similar discs were taken, perforated in the centre by a hole a quarter of an inch in diameter.

Corresponding with this hole in the lower disc, a cut half an inch long was made completely through the membrane. The retraction of the membrane converted this slit into an oval hole, and in this way, when the discs were placed in apposition, a free communication was provided between the air and the space between them. I found, however, that the same weight was still required—viz., 325 grammes—to separate them. A similar slit was made next in the upper disc and membrane as well, but this, too, made but a few grammes' difference. The result of these observations is to prove that there is some force, other than atmospheric pressure, by which these two smooth surfaces are held together, and without using the term in too technical a sense, I may speak of it as cohesion.

The next point to be determined was this—Was this cohesion greater or less than the elasticity of the lungs?

Accepting, for the sake of the calculation, the higher estimate made by Donders—viz., 7 millimetres of mercury—it was found that a column of mercury of this height, with a circular base of 5 centimetres in diameter, weighed 185·6 grammes. The weight required to separate the discs was 325 grammes, so that there was an excess of about 140 grammes in favour of

[1] Bradshawe Lecture, *Lancet*, Aug. 1887.

the force which kept the discs together. If this weight be reckoned out in terms of mercury, it is equivalent to 12·5 millimetres, as opposed to 7 millimetres, giving thus an excess of 5·5 millimetres.

Having thus roughly established the correctness of the theory, I endeavoured next so to modify the experiment as to imitate somewhat more closely the conditions which exist in nature. I took a bell-jar, the mouth of which was about 3 inches in diameter. The top was bored so as to provide for the insertion of a perforated cork, into which a glass tube could be fitted, and communication made in this way between the external air and the interior of the jar. Over the mouth two pieces of stomach were stretched, with the peritoneal surfaces in contact. By exhausting the air the membranes were made to bulge inwards ; they remained, of course, in contact. A slit was now carefully cut through the outer membrane, forming an oval hole about half an inch long and a quarter of an inch wide. This hole provided for the access of air between the membranes, just as in the case of the discs. When the air was now exhausted the two membranes still remained in contact as at first, and it was only after considerable bulging that they separated and air passed between them. A manometer was now connected with the interior of the jar, so that the pressure could be measured. By means of a special modification of the mercury manometer the reading could be converted at once into its equivalent in inches of water.

7·0	millimetres of mercury,	3·8	inches of water.
12·5	,, ,,	6·8	,, ,,

The jar was now exhausted until the negative pressure indicated was 3·8 inches of water. At this pressure, the equivalent of the elastic tension of the lung, the membranes remained in contact. The exhaustion was continued until a pressure of 6·8 inches of water was reached, but still the surfaces remained in contact. The pressure could even be slowly still further reduced to 10 or 12 inches of water before the surfaces separated and air passed between them. When the pressure was relieved they again passed into contact, often so completely that the original experiment could be repeated. More often, however, unless the membranes had been very carefully stretched, a few small bubbles of air remained, which had to be pressed out with the finger to make the contact perfect.

It is not to be wondered at that with the bell-jar experiment higher values were obtained for the force of cohesion than with the discs. It is clear that although I had discs of five centimetres in diameter covered with membrane, the actual surface of contact was something less than this, and the results obtained were therefore below the real value. While this value was in all probability too low, I accepted for the purpose of comparison the highest estimate of the elasticity of the lungs, so that on both accounts the difference arrived at was less than the real difference.

With the bell-jar experiment certain precautions are necessary. The two pieces of membrane must be such as will stretch uniformly. This makes the parietal pleura itself not a very suitable membrane for experiment. It is much thinner in the parts corresponding with the ribs than it is in the parts corresponding with the intercostal spaces. The thinner parts yield most on exhaustion of the jar, so that the surface is thrown into furrows, and the tension on bulging differs in consequence in different parts. This affects greatly the result. The stomach is more suitable ; but even with this care must be taken in the selection of the parts used, for when stretched the large blood vessels act as bands, and, if they be included, folds are also produced.

The membranes also must be so stretched as to have as nearly as possible the same tension in each. If this be done the surface may remain in contact at much greater negative pressures ; it may even be at 2 inches of mercury before separation is effected. This result was so much in excess of what I anticipated that I sought for some explanation of it. While the membranes were on the stretch, therefore, at this pressure I gently raised the edges of the slit with a blunt probe, so as to admit a little air between the membranes here, and I found then that the two layers separated at once. I repeated the experiment at different pressures. As the pressure was reduced more and more, the ease with which air passed between the layers when the edges were raised became less and less, until, when the pressure was below 12·5 millimetres of mercury —that is, below the pressure at which the force of cohesion had been originally estimated—in spite of the edges being freely separated by the probe, the membranes did not separate, but directly the outer layer, which had been lifted up, was allowed to fall, it passed again into close contact with the other, and the air which had been admitted was forced out. This experiment appears to me to be conclusive.

The figures above are actual numbers obtained in particular experiments. The numbers differ a good deal in different observations, their variations depending upon differences in the stretching of the membranes, and in the conditions of their surfaces at the time of experiment. Without laying, therefore, any special stress upon the actual values obtained, these observations establish, I consider, the existence of a force between the pleural surfaces much in excess of the elasticity of the lungs, and sufficient, therefore, to maintain the lungs in apposition with the walls of the thorax even when air has free access to the pleural cavity.

Pneumothorax can therefore be no longer regarded as a condition to which there is an inherent tendency in the healthy body, but, on the contrary, as a condition brought about by the forcible separation of the pleural surfaces, and in this respect exactly analogous to the distension of the subcutaneous tissue which occurs in surgical emphysema; and considering the frequency of subcutaneous emphysema and the rarity of pneumothorax in simple fracture of the ribs, to which reference has been made, it would seem to follow as a corollary that the force required to distend the subcutaneous tissue must be less than that required to separate the layers of the pleura. Pneumothorax, therefore, so far from being, as it is commonly regarded, a passive process, and inspiratory in origin, is really expiratory in its initial stage, and requires an active force to produce it; and surgical emphysema, so far from implying, as Fagge asserts, the necessary antecedence of pneumothorax, may in reality be a protection against it, the air making its way in the direction of least resistance, beneath the skin rather than between the pleural surfaces. It is evident, further, that where the pleura is laid freely open, so that the air passes away without hindrance on expiration, the liability to pneumothorax will be still further reduced, for the expiratory pressure requisite to overcome the cohesion of the pleural surfaces will be absent.

By means of the discs I was able to investigate some of the conditions which modify the force of this cohesion. In the first place, the surfaces must be absolutely smooth, for any roughness prevents close contact. This is one of the reasons why stomach well stretched over the discs is more suitable than pleura, the mucous membrane forming a cushion into which any little unevenness will sink. I have frequently found that the same pieces of pleura which would not give good results when spread directly on the discs, answered admirably as soon as they were stretched with a piece of stomach beneath them.

The surfaces in contact must be neither dry nor wet, but simply moist, as are the normal surfaces of the pleura. If dry, they will not cohere at all; if wet, the force of cohesion is greatly reduced. The cohesion is, however, not between the membrane and the surface of the fluid, for a disc floated on water, blood, or serum requires but little force to raise it out. What is essential appears to be an extremely fine film of fluid, a capillary film, if I may call it so, between the two surfaces. It may be that the cohesion varies with the nature of the fluid; but so far as I have gone at present, I do not find, with my rough methods of investigation, any perceptible difference between serum, blood, or water. The ready abolition of this cohesion by the presence of even a small excess of fluid is important, for there will be on this account less resistance to the entrance of fluid than of air into the pleura. This force of cohesion has a general bearing in physiology, for it will exist wherever serous surfaces are in close contact; in the peritoneum, therefore, and in the pericardium as well as in the pleura. In the peritoneum its tendency will be to keep the intestines partly distended, and in this way it will diminish resistance to the passage of its contents along the tube. It will be perhaps in the joints that one of the most interesting illustrations of its usefulness will be found, for it is compatible with the freest movement possible of the surfaces upon each other. It may perhaps prove to be one of the most important means by which the head of the femur is kept in the acetabulum, but it will come also into play even in the hinge-joints. Since the force appears to be directly proportional to the extent of surface of contact, it will continue to be the same so long as the extent of surface of contact does not vary, and in the hinge-joints, therefore, so long as this is the case, the same amount of force will be exerted whatever be the position of the joint to keep the ends of the bones in place. The effect of excess of fluid in diminishing or even abolishing this force is well illustrated by the looseness which develops in the joints when effusion takes place into them.

But to return to the pleura, with which we are now more especially concerned. The existence of this force of cohesion is sufficient to explain satisfactorily many of the clinical difficulties which present themselves and which led me to institute these investigations.

THE SYMPTOMS.—*THE PHYSIOLOGICAL CONSEQUENCES OF PNEUMOTHORAX.*—As soon as the air has gained access to the pleura, the elasticity of the lung comes into play and leads to its collapse. Whether

the elasticity of the lung alone be sufficient, as is often stated, to produce complete collapse or not, is a question which is of more theoretical than practical importance in pneumothorax; for in most cases in the early stages the air enters the pleura on inspiration with greater ease than it can escape on expiration, and thus there is added to the forces tending to produce complete collapse of the lung the compression of it during expiration. The result is that in a very short time the lung becomes completely collapsed and airless.

If the lung be free from adhesions, it lies shrunken about its root and flattened against the vertebræ. If, however, there be adhesions, it will contract in an irregular fashion. If the adhesions be at the apex, as they commonly are, the contraction of the lung takes place in a more upward direction. If the adhesions be in front, along the sternum, the lung may be flattened sideways, and lie like a pancake between the sternum and the spine. In such a case as this the heart and mediastinum may be fixed in their usual place, and no displacement of organs occur. When the adhesions are limited, the lung in the corresponding part may be drawn out into a long band, which I have seen stretched across the pleura and measure some inches in length.

These adhesions, and the peculiarities of contraction of the lung to which they lead, it is important to bear in mind, as they may explain some of the irregular physical signs occasionally met with.

The effect of the collapse of the lung on the affected side is to render it absolutely useless for the purposes of respiration.

Displacement of Organs.—The heart and mediastinum are firmly fixed to the spine behind, and but loosely attached to the sternum in front, so that they are capable of considerable displacement, much as a door swings on its hinges. The mediastinum occupies its usual place in the middle line because of the elasticity of the two lungs which balance it on either side. If, then, the elastic traction of one lung be abolished, as it is in pneumothorax, that of the other side, being unopposed, comes into play and drags the heart and mediastinum over on to the sound side. This displacement is the necessary consequence of pneumothorax, if the mediastinum be free to move: it can only be absent under two conditions; first, when the mediastinum is fixed by adhesions; secondly, when the other lung has also lost its elasticity or power of retraction.

Although, as Douglas Powell showed, elastic contractility of the sound lung is of itself sufficient to account for the maximum displacement of the heart and mediastinum, still it is supplemented, at any rate in most cases, by expiratory compression, as already described.

The effect upon the opposite lung in a case of pneumothorax, therefore, is considerable; for whereas it formerly filled that side of the thorax, it now occupies a space so much smaller by the room that the displaced heart and mediastinum take up, which may be roughly estimated at perhaps a quarter of it.

If, for the sake of argument, we assume that the two lungs may be represented in capacity by 5 for the right and 4 for the left, we have a reduction in the respiratory capacity which we may express as follows:—

For right-sided pneumothorax we have the total respiratory capacity of the two lungs, less the respiratory capacity of the right and a quarter of the left $= 9 - (5 + 1) = 3$.
For left-sided pneumothorax we have, *mutatis mutandis*, $9 - (4 + 1\frac{1}{4}) = 3\frac{3}{4}$.

Thus the total respiratory capacity of the lungs is reduced considerably more than a half; it may even be as much as two-thirds, and this on the assumption that the opposite lung is healthy. Where that lung is also the seat of disease, the reduction in respiratory capacity will be still greater.

The physiological or functional capacity of the lungs is, however, still further reduced, for other reasons. The collapse of the one lung as a whole is associated with a proportionate collapse of the vesicles of the other; for they not only contain a smaller volume of air, but also offer a smaller aërating surface on which the blood vessels are exposed to the air.

To some extent an attempt is made to compensate for these defects by increase in the depth and number of respiratory movements, but very ineffectually; while the inspiratory forces are greatly diminished by the fact that only one side of the diaphragm has any effective action at all.

These defects are grave enough; but there is still another, which is perhaps still more important than either, viz., the sudden embarrassment of the circulation which pneumothorax causes.

In the first place, the one lung left is suddenly called upon to do the duty of two. The right heart goes on attempting to pump its usual amount of blood out, but there is only one lung for it to pass into. Unless the blood can pass through the lungs with at least twice the normal rapidity (and this is clearly impossible), the blood must accumulate in the lungs, the vessels will become distended and the lungs congested. As the result of this, the free exchange of gases in the vesicles of the lung between the blood and the air is impaired, and thus the passage of the blood through the lung capillaries is rendered more difficult than before. The heart endeavours to overcome this obstruction by beating more rapidly and more forcibly; but soon it fails. Then the difficulties of the circulation are still further increased, and the already failing heart becomes still more distended.

So long as the remaining lung and the heart are able to cope with the extra work thus thrown upon them, the lung will yield no physical signs; but so soon as the work becomes too much for them, the congestion of the lung will show itself in the usual way with the signs of bronchitis, and sometimes with hæmoptysis; while the failure of the right heart betrays itself by increasing dilatation, irregularity and feebleness of action. Thus the end may come in two ways, either from congestion of the lungs or from over-distension of the heart. This is the condition appropriately named by Wintrich "*Insuffisance aiguë du poumon.*"

In the causation of the urgent symptoms, time is a very important factor. It is not so much that one lung is helpless and the other has to do double duty, but that the change is so sudden that neither lungs nor heart have had time to adjust themselves to the altered conditions. Although, in pleuritic effusion, one lung may be as completely collapsed and useless, and the displacement of organs on to the opposite side may be as great, as in pneumothorax, still such urgent symptoms are rarely seen. The difference is due to the fact that in cases of pleuritic effusion the changes have been more or less gradual, or, at any rate, not nearly so sudden. At the same time, even in pneumothorax, when the patient does not succumb at once, the heart and lungs, if they be healthy, may adjust themselves to the altered conditions, and the urgency of the symptoms then gradually subside.

The great danger of pneumothorax is during the first hour or so after its occurrence, and the immediate prognosis improves with every hour that life is prolonged.

The urgency of the symptoms of pneumothorax depends, therefore, principally upon the suddenness of the change in the respiratory conditions, and next upon the strength of the heart and upon the condition of the opposite lung.

The maximum change is met with in cases where the previous health has been good, and where the lungs have not been obviously diseased. Accordingly,

the most intense symptoms in pneumothorax are met with in those who have been previously apparently in good health; while, on the other hand, in some cases of well-marked phthisis the symptoms may be so slight that pneumothorax may be overlooked. This apparent paradox is intelligible if it is looked at from the point of view of the amount of change in the respiratory conditions which is produced in the two cases. Thus where the patient has been in apparently good health previously and the lungs are but little diseased, the displacement of organs and the consequent collapse of the opposite lung reach their maximum, while the congestion of the opposite lung is extreme, the patient being full blooded and all the nutrition processes in full activity. On the other hand, in most cases of phthisis the patient has been ill for some time, the amount of blood in the body and the consequent congestion of the opposite lung is much less; moreover, one lung has probably for a long time been doing little work, and it may really make but little difference in the respiratory conditions whether the one lung is useless on account of phthisis, or because it is collapsed as the result of pneumothorax. At the same time, if the lung on the opposite side be also extensively diseased, the reduction in respiratory capacity may be so great as to be incompatible with life. Yet it is extraordinary how little lung seems necessary in some cases for mere existence, so that even in advanced cases of phthisis the occurrence of pneumothorax may be marked by hardly any symptoms.

When the symptoms are urgent, other things being equal, the strength of the heart (which also depends greatly upon the general strength of the patient) will largely influence its power to deal with the altered conditions.

So we find that in some cases of advanced phthisis the occurrence of pneumothorax may lead, not to the ordinary symptoms of pneumothorax, but to cardiac syncope, and this may end in sudden death, or a long collapse from which the patient may never rally.

SYMPTOMS.—Onset.—The onset of pneumothorax is sudden, and often without obvious cause.

The patient is seized all at once with pain in the side and shortness of breath. The difficulty in breathing rapidly increases and in a few minutes becomes extreme. The patient is now found sitting up, panting and gasping for breath, rapidly becoming more and more cyanosed, and with an expression of the greatest anxiety and distress; unable to speak, or, at any rate, to utter more than a syllable or two at a time; the whole body bathed in perspiration and the extremities cold. There may be a little cough, which, like speaking, adds greatly to the suffering. The mental distress is great, for the patient looks and feels as if about to die.

The symptoms rapidly grow worse, and it is evident that unless relief be given the patient will die. A needle is inserted into the side; air escapes and the breathing is relieved; the needle is removed, but the air again accumulates and the symptoms become once more urgent. A second time, and it may be a third, relief is given by paracentesis; but often the relief is but temporary and the symptoms soon become as bad as ever; the opposite lung becomes congested, the right heart dilated, and the patient dies, it may be within an hour or two of the commencement of the attack.

If the result be not to be so immediately fatal, the interval between the tappings increases, the dyspnœa gradually becomes less severe, and in twenty-four hours or so the extreme urgency of the symptoms passes away.

Dyspnœa.—The dyspnœa depends upon several factors, as already shown.

1. Upon the rapid collapse of the affected lung.
2. Upon the partial collapse of the opposite lung caused by the displacement of the organs.
3. Upon the consequent congestion of the opposite lung.

Although it is the rule for pneumothorax to be ushered in by the grave and urgent dyspnœa described, still it is not always so, and the absence of such acute symptoms is not conclusive against the presence of pneumothorax. In some cases, indeed, there may be little to suggest what has occurred, and the pneumothorax may be discovered only by physical examination, when there has been hardly any appreciable dyspnœa to draw attention to the chest. These cases are usually called *latent* or *insidious pneumothorax*, and will be dealt with by-and-bye.

If, as in some of these phthisical cases, there be widespread adhesions, the collapse of the lung on the affected side may be less, and it is also possible that the displacement of organs may be prevented; these cases are often spoken of as *partial pneumothorax*.

Where there is extensive disease of the lung on the affected side, it may make little difference to the patient whether the one lung is useless because it is infiltrated with tubercle, or because it is collapsed as the result of the pneumothorax, and the symptoms, therefore, may be slight, or even entirely absent.

If, however, the opposite lung be also much diseased, the dyspnœa must necessarily be extreme, especially if there be much displacement of organs, for the reason that there is so little lung left for the performance of respiratory purposes.

Where pneumothorax occurs in the course of advanced phthisis, there is one other cause of dyspnœa which deserves to be mentioned, though it is not usually referred to. Where the lung on the affected side contains many cavities, the secretion contained in them may be suddenly expelled into the air-tubes as the lung collapses, and if not immediately coughed up may very seriously aggravate the dyspnœa. I have seen a patient all but suffocated in this way, and in the two cases in which death occurred in twenty minutes and thirty minutes respectively from the time of onset, it was largely due to this cause.

Pain.—The sensation usually experienced is that of a sharp stabbing or tearing pain, and is often described by the patient "as if something had suddenly given way in the chest." It is usually of short duration and not very severe, or, at any rate, not severe for long. It is commonly felt in the mid-lateral region, *i.e.*, in the axillary region; but it may be referred to the upper part of the chest in front or under the breast. Occasionally it is referred to the spine, to the angle of the scapula, or even to the abdomen, and it may radiate round the chest.

In one case, subsequently recorded, the pain was so severe that the patient could not be prevented from shrieking out, but the dyspnœa was not grave. In some cases the initial symptom complained of is not that of pain, but of some other abnormal sensation, such as cold water running down the side or of air rushing into it.

In the later stages the usual sensation is that of distension or tightness, which, though distressing, can hardly be called pain.

Collapse.—The occurrence of pneumothorax is sometimes marked, not by pain or dyspnœa, but by a sudden attack of faintness or collapse, upon which dyspnœa follows as soon as the patient rallies. Occasionally the collapse may be fatal. Pneumothorax may thus be the cause of sudden death, an extremely rare occurrence, of which I have never seen an instance myself. Lebert describes a case of the kind in a medical man of 28 years of age. Though I have not seen death from shock owing to perforation of the pleura, I have seen it follow perforation of the peritoneum. In this case the rupture of a hydatid of the liver caused death, the patient, a previously healthy young man, falling down suddenly in the street, and being picked up dead.

The Temperature.—This presents nothing specially noteworthy. In itself, pneumothorax need not affect the temperature at all. The onset, it is true, is usually attended with a drop in the temperature, which may be considerable if there be much shock or collapse.

If any elevation of temperature be present, it is due not to the pneumothorax as such, but to the original disease which has caused it, *e.g.*, phthisis, or to the complications to which it has led, *e.g.*, pleuritic effusion.

Where the temperature has been previously raised, as in a case of phthisis, the onset of pneumothorax may be marked by the usual drop as stated, and it may be some little time before the previous level of temperature is reached again. This fact has been used as a strong argument in favour of what I believe to be a fallacious theory, viz., that pneumothorax, or, to put it more generally, the collapse and compression of the lung, to which pneumothorax leads and to which, of course, pleuritic effusions similarly lead, check the progress of tubercle. Admitting the fact that the pneumothorax may be followed by a diminution of fever, it may be permitted to question the explanations that this is due to a check in the development of tubercle in the compressed lung. To this theory there are many objections—and as it involves questions of practice, it will be again referred to, under Treatment. For the present, the objections may be simply stated:

1. That the theory has more exceptions than proofs.
2. That the occurrence of pneumothorax is often followed by a very rapid progress of tubercular mischief on the opposite lung.
3. That the relief of the collapse by operation is rarely followed by progress in tubercle, as it would be likely to be if the theory were true.
4. That recent tubercles, apparently of formation subsequent to the occurrences of pneumothorax, may be present in the collapsed lung.

The explanation which I should give of the fact is this. The fever of phthisis is in great part due to septic absorption from cavities which are the seat of secondary infection with pyogenic organisms, and is the same in character as that due to an abscess or pent-up pus. The collapse of the lung may act like the opening of an abscess, evacuate the contents of the cavities, and be therefore followed by a fall of temperature. If this explanation be correct, the diminution of fever has nothing whatever to do with the rate of progress in the tubercular mischief.

The pulse varies greatly according to the general condition of the patient and the severity of the dyspnœa. Under any circumstances, the respirations are likely to be more affected than the pulse, and accordingly the pulse-respiration ratio becomes perverted even to almost as marked a degree as in pneumonia, and may be as 3, or even 2, to 1. If the onset of pneumothorax has been attended with shock or collapse, the pulse will be small, irregular, or perhaps hardly to be felt at the wrist, as in a patient fainting or collapsed from other causes. If the dyspnœa be extreme, and suffocation imminent, the pulse shows the embarrassment of the heart and of the circulation through the lung, it becomes irregular in force and frequency, fluttering, and of low tension, and though at first laboured and not much accelerated, it may become later very rapid and feeble.

When the dyspnœa has passed off, the pulse recovers itself quicker than the respirations, so that the perverted pulse-respiration ratio may still continue for some time. It may then show no peculiarities other than would be met with in any person of feeble health, *i.e.*, it is of low tension and easily disturbed, in rate and power, by slight causes.

Position of the Patient.—As long as there is urgent dyspnœa, the patient will sit upright (orthopnœa), or lie in the semi-recumbent position with the shoulders raised. When the urgency of the dyspnœa is past, the patient will lie in any position, as may be most comfortable; this varies much, but is usually upon the affected side, with the object, no doubt, of giving the opposite lung full play.

PHYSICAL SIGNS.—The characteristic physical signs are distension of the side, displacement of organs, and tympanitic percussion; to which may be added the bell-sound, and if fluid be present, succussion; while in most cases the breath- and voice-sounds are absent.

Inspection.—The respirations may number 40, 50, or more in the minute, but there is a strong contrast between their number and their depth, as there is also between the shape and movements of the two sides.

The shape of the chest.—The affected side is greatly distended, the shoulder raised, the sternum thrust forward, and the intercostal spaces widened. This distension is not limited to the affected side only, but involves to some extent the other also; for the elastic traction of the lungs upon the ribs, which tends to keep the side somewhat smaller than it would otherwise be, is removed or diminished, in consequence of the displacement of organs on to the sound side. In spite of this, measurement of the two sides may show a difference of an inch or so in the circumferences.

This position is often described as the "maximum inspiratory position"; but it may exceed anything which can be intentionally imitated in health.

If the intra-pleural tension be low, the distension may be less marked, and if there be a large opening into the lung, so that there is no increase of tension at all, the distension may be entirely absent or the side may even be contracted somewhat, much as it is after it has been opened for empyema; so that, although distension of the side is an important sign of pneumothorax, its absence does not count for much against that diagnosis.

The respiratory movements.—On the affected side the respiratory movements are absent; in other words, the distension is fixed, for there is no expiratory retraction. If, however, there be a free opening through the lung, the movements are present, or may even be exaggerated, though they have but little effect upon the lung, which, if it expand at all, is forced out only during expiration, rather than sucked out by inspiration.

On the opposite side the movements are shallow, and the respiratory excursion is small, for though the side attains its maximum inspiratory expansion, it fails to retract to the extent it should.

The movements of the diaphragm are also deficient on expiration on both sides, but especially on the affected side.

The *rapidity of breathing* varies with the dyspnœa, and if dyspnœa be absent, or, in the subsequent stages, when it has passed off, the respirations may be but little, if at all, accelerated except on exertion.

The *superficial veins over the affected side*, and also on the corresponding side of the neck and down the arm, are sometimes found dilated. This is rightly referred to obstruction of the intra-thoracic veins. It is, however, a rare phenomenon, and only met with when there is high intra-pleural pressure, especially when there is a considerable effusion, and the conditions have existed for some time. It is, therefore, not so often met with now that pneumothorax is recognised earlier, and treated more actively.

In the same way *œdema* may be seen; not a local œdema due to the pointing of pus, as may be met with in any neglected pyo-pneumothorax, but

a general œdema of the whole side, the result of the venous obstruction just referred to.

The *impulse of the heart* is far out of its proper place. On the left side this will be the apex of the heart, and may be in the anterior axillary line; on the right side the impulse is that of the right auricle or ventricle, and may be seen in the right nipple line, or even an inch or so beyond it.

Palpation.—The *impulse of the heart* may be felt as well as seen in its new position.

The *vocal vibrations* are usually completely absent over the whole affected side, but may be sometimes indistinctly felt. On the opposite side, if there be much congestion of the lungs, wheezing may be felt, but if not, there is nothing abnormal on palpation.

Percussion—Displacement of Organs.—The mechanism of displacement has been already considered. It is due chiefly to the elastic retraction of the lung on the sound side, but it is supplemented on the affected side by the increased pressure on expiration, and by the general rise of intra-pleural pressure when fluid forms. As the result of these causes the heart and pleura are displaced far over on to the opposite side; while the diaphragm falls, and carries the organs in relation with it downward into the abdomen.

The displacement is chiefly determined by means of percussion. The side yields a tympanitic note, which is obtained in all directions as far as the pleura extends. Thus it may reach an inch or two beyond the sternum on to the opposite side, to the costal arch below or even somewhat beyond it.

The effects of displacement differ a good deal on the two sides, and it will be convenient to consider them separately; for with right pneumothorax it is the liver that is chiefly affected, and with left pneumothorax the heart.

With Pneumothorax of the Right Side.—The heart is seen and felt beating 2 inches or more outside the left nipple line, and the area of cardiac dulness is correspondingly displaced, the right border being found an inch, it may be, to the left of the sternum.

The chief displacement on this side is in the position of the liver, for this is not only pushed downwards, but is curiously rotated. Percussion yields a resonant, tympanitic note right down to the costal arch. Usually at this place the hepatic dulness makes itself evident; but in some cases, where the diaphragm is so far depressed as to be convex towards the abdomen, there may be a zone of resonance 1 to 2 inches in width, between the edge of the ribs and the upper border of the liver; this, however, is but very rarely met with.

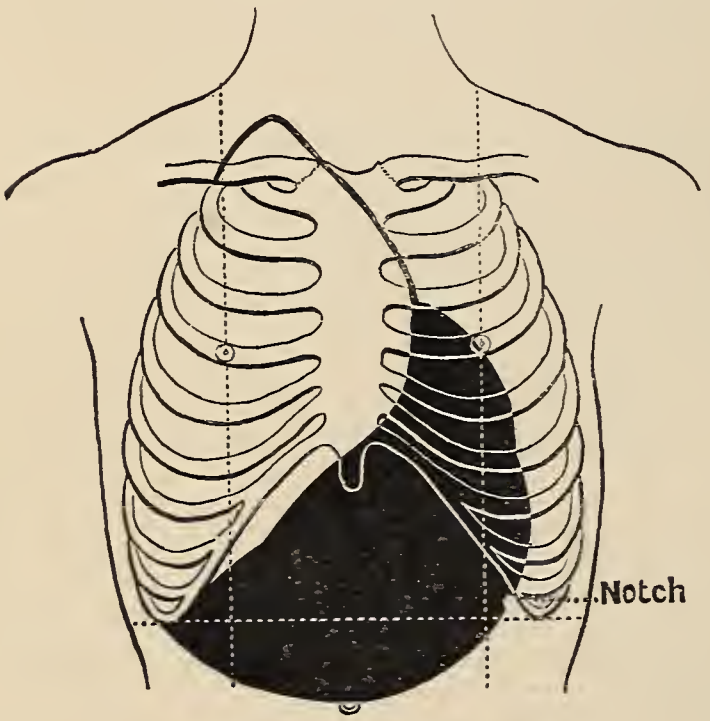


Fig. 157.

Diagram of right-sided pneumothorax, showing the great displacement and rotation of the liver, the notch being nearly under the costal arch on the left side, and the gall bladder to the left of the umbilicus.

As the right lobe of the liver is so much displaced, while the left retains its ordinary position in relation with the heart, or at the most is displaced an inch

to the left, it is evident that the organ will be greatly rotated or twisted. Thus the lower border of the liver may reach nearly to the iliac fossa, and from thence ascend in a curved line slightly below or through the umbilicus nearly up to the position which the apex of the heart occupies. The notch is usually found either immediately beneath the left costal arch or an inch or so away from it; while the gall-bladder may be either in the middle line or slightly to the left of it. The spleen is in its normal position, but the area of stomach resonance between the liver and the spleen is, of course, much reduced.

Extreme as the displacement of the liver is, I do not know that it ever produces any disturbance in its function.

With Pneumothorax of the Left Side.—Here it is the heart that is chiefly displaced. The descent of the diaphragm carries down with it below the costal arch, the stomach, the spleen, and, to some extent, the left lobe of the liver.

The spleen may be felt quite distinctly below the ribs; the left lobe of the liver is thrust slightly down, but beyond this the liver retains its normal position, so that its upper border remains in its usual site on the right side, viz., the upper border of the sixth rib in the nipple line.

The displacement of the heart is remarkable, for the impulse may be seen and felt even 2 inches outside the right nipple line. The impulse felt here was formerly stated to be the apex beat, for it was supposed that the heart was fixed by the large vessels at the base, and that when it was displaced a distinct rotation took place, so that the apex moved from its normal position on the left side to a similar position on the right side. *Post-mortem* observation proves this to be quite incorrect, and what really happens is that the heart moves bodily over with the mediastinum, retaining approximately its normal relation to the diaphragm, and suffering no rotation; the impulse felt to the right of the sternum being that of the right auricle, while the apex, even with the maximum displacement of the heart, lies either beneath the lower part of the sternum or hardly beyond it. This the two accompanying diagrams prove; the one showing the surface markings during life, and the other the actual position of the organs on *post-mortem* examination.

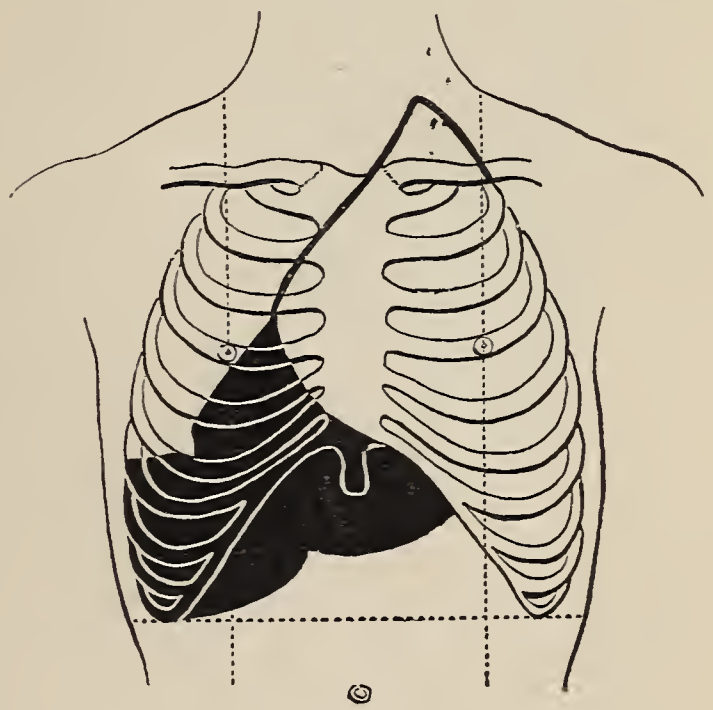


Fig. 158.
Diagram of left-sided pneumothorax.

The displacements are the same as that which is met with in large effusions into the pleura, and in neither case does the displacement, great as it is, produce any marked effect upon the heart's action.

Murmurs due to the displacement are described; but they are certainly as rare in pneumothorax as is pleuritic effusion. I do not remember ever to have seen an instance of them myself, nor have I seen any evidence in pneumothorax, any more than in pleuritic effusion, of that kinking of the vena cava which has been described as the cause of sudden death in some of these cases.

As already stated, percussion yields the same tympanitic note over the whole affected side, the only possible exceptions being over those parts where the lung may happen to be adherent or collapsed, or where fluid is present in the pleura.

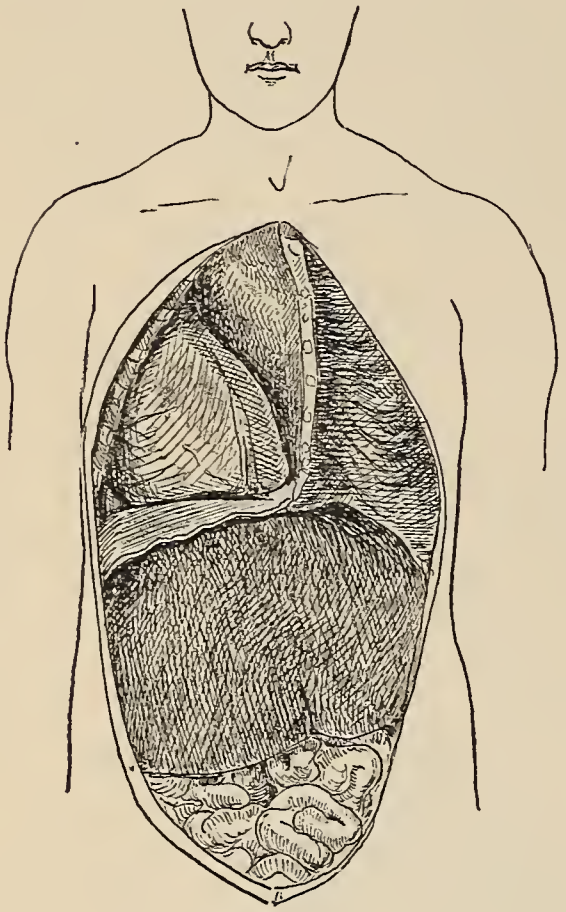


Fig. 159.

Diagram to show the mode of displacement of the heart in pneumothorax of the left side. The chest was opened by an incision down the middle line of the sternum, and the right half of the front wall removed. At the upper part is seen the left pleura, greatly distended, and forming a round, tense protrusion, extending far over on to the right side and reaching the tip of the 2nd rib. The pericardium has been opened and the heart exposed. The heart is seen to be simply displaced to the right, the apex lying beneath the right edge of the sternum. The front part of the heart, as seen, consists of the right ventricle almost entirely. There is no twisting of the heart on its axis; but the mediastinal structures, bronchi and great vessels are all displaced to the right side, the trachea even passing nearly behind the right sterno-clavicular joint. The liver is greatly depressed but not much rotated.

If the lung be adherent at the apex, the percussion note may be impaired; but it becomes "boxy" rather than absolutely dull, for the resonance from the pleural cavity below is transmitted through the solid lung or along the chest walls, and there may be even considerable adhesions at the apex without any impairment of the note at all. In the interscapular space behind, also, the note, theoretically, may be impaired if the lung be collapsed, as it commonly is, round its root; but in this case also any dulness that may be produced is usually masked by the surrounding hyper-resonance.

Even when fluid is present in the pleura, it may be in considerable amount and yet yield no dulness. The reason of this in most cases is, that when the patient is in the semi-recumbent position, or sitting up for the purposes of examination, the diaphragm takes the shape of a saucer, so that a good deal of fluid may lie in it, deep and out of reach, and thus not affect the percussion-note.

Hence it is that succussion may often be easily obtained when percussion gives no evidence of fluid. When the amount of fluid is large enough to give dulness, the level is then found to change readily with position, and with very much greater rapidity than is the case in simple pleural effusion.

The percussion note is said to change its pitch according as the mouth is open or shut. I have often tried, but have never succeeded in satisfying myself, that this statement is correct.

It is also stated that when the intra-pleural tension is high, the percussion-note may lose its tympanitic character and become impaired, or even dull. I have never seen anything at the bedside to justify this statement, and the experimental observations that I have made go far to disprove it, and have led me to reject it.

Auscultation.—The voice- and breath-sounds are usually absent, just as they are in an effusion of fluid, except, it may be, in places where the lung is still in contact with the chest walls. Thus the breathing may be bronchial or amphoric in the interscapular space behind, where the lung lies collapsed near the spine. At the apex also, when the lung is adherent, the breath sounds may be exaggerated. This, how-

ever, is not nearly as frequent as might be anticipated, the explanation being, I suppose, that the lung here is so completely collapsed that even the air-tubes contain but little air, and the breath- and voice-sounds are not transmitted.

Although, as stated, it is the rule for the voice- and breath-sounds to be absent over a pneumothorax, still there are cases in which they are greatly exaggerated, the voice sounds being distinctly heard, and the breathing being bronchial or amphoric in character.

Of this, two explanations are usually offered.

1. The first, that in such cases there is a wide opening into the lungs, allowing air to pass freely in and out of the pleura, so that the voice- and breath-sounds are transmitted directly to the air in the pleura. Pneumothorax then offers practically the same conditions as a large pulmonary cavity, and yields similar auscultatory sounds.

This explanation, however, can, at best, only be partly true; for even when the pulmonary opening is free, amphoric breathing is not always heard, and sometimes no breath sounds at all are audible; while, on the other hand, amphoric breathing may be present when it was obvious during life that the opening was closed, a fact which *post-mortem* examination proved.

Thus I remember a man with pneumothorax, in whom amphoric breathing was at first absent, but developed subsequently, and that at a time when it was evident from the contraction of the side and the return of the organs to their normal places that the air was being rapidly absorbed from the pleura, and that, therefore, no free opening could exist through the lung. As the air continued to be absorbed, the amphoric breathing ultimately became fainter and fainter, and finally disappeared, its place being taken by the normal respiratory murmur.

2. The other explanation is that the amphoric breathing is the effect of consonance, the sounds—produced in the collapsed lung, and often heard behind even when there are no respiratory sounds to be heard elsewhere—being taken up and reinforced by the air contained in the pleura. This explanation also seems unsatisfactory; for if it be adequate to explain the less common cases in which amphoric breathing occurs, it does not explain why this is more frequently absent. Consonance certainly cannot explain the bronchial or amphoric breathing which is present in some instances of fluid effusion. I think it must be admitted that there is no really satisfactory explanation of the phenomenon, which will fit all cases.

The heart sounds are sometimes heard loudly over a wide area in pneumothorax, and may have a somewhat amphoric character.

This is also explained as the result of consonance, but it is more often absent than present.

Whatever the explanation given, it must be recognised clinically that there are, in respect of physical signs, two groups of pneumothorax; the first (the common form) that in which both the voice- and the breath-sounds are absent, the physical signs being like those of pleuritic effusion, except that the side is resonant instead of dull; the second and rarer form, that in which both voice- and breath-sounds are audible and usually exaggerated, so that they have a bronchial, or it may be an amphoric, character.

Two phenomena, which auscultation yields, are very characteristic, viz., the bell sounds and succussion, to which may be added perhaps a third, viz., the metallic tinkle or echo. Though very suggestive, they are not absolutely pathognomonic of pneumothorax, for they may be heard in a large pulmonary cavity, and the two former also over in a distended stomach.

The Bell Sound.—If a coin be placed flat upon the walls of the thorax in front, and tapped with another, while the chest is auscultated in the axilla or behind, under ordinary circumstances nothing is heard, except a dull, metallic tap, like that produced by striking the two coins together upon the hand; but in pneumothorax the sound acquires a clear, ringing character, much like that obtained on striking a metal-bell or porcelain-bowl. This is called the "bell sound."

Though known to Laennec, it was especially studied and described by Trousseau under the name of "bruit d'airain."

The bell sound, though very suggestive of pneumothorax, is not quite pathognomonic, for it may be heard over a dilated stomach, and occasionally over a large cavity in the lung.

The conditions upon which the phenomenon depend are not clear; but the cavity must be of considerable dimensions, and, I think, it must have smooth walls. The tension of the air within the cavity cannot have much to do with it, for it may be heard in pneumothorax where the intra-pleural tension is high, *e.g.*, several inches of water, as well as in cases where the tension is not above that of the atmosphere. It has been heard in the corpse when it was absent during life, and it was this fact that led Traube to suggest the view that it was the result of diminished tension. That the matter is not as simple as it may at first sight seem, is clear from the fact that though generally present in pneumothorax, it is sometimes absent, and that it is not heard everywhere over the side, but only in certain places. Trousseau maintained that, in order to elicit it, the part auscultated should be directly opposite to that on which the coins were placed. This, however, is not the case, and it is not possible, before trying, to say in what position the bell sound will be best obtained.

In places where the lung is adherent to the chest walls, the bell sound will not be heard; but it may be absent in parts where there are no adhesions to explain it. On the other hand, especially in partial pneumothorax, or where the lung has in great part expanded after an ordinary pneumothorax, it is possible, sometimes, by means of the bell sound, to mark out fairly definitely the limits of the air-containing cavity.

Succussion.—This phenomenon, familiar to Hippocrates, is the splashing sound produced by suddenly shaking fluid in an air-containing cavity. There are three conditions essential to its production, viz.:

1. That the cavity should be of fairly large size.
2. That it should contain both air and fluid; and
3. That the fluid should be splashable, *i.e.*, should not be so viscid that it could not be made to splash by any shaking to which the patient could be fairly subjected.

Succussion can be produced in the stomach of any one after hastily drinking a large quantity of fluid, and in some cases of dilated stomach it may not be easy at first to decide whether the succussion is produced in the stomach or in the left pleural cavity.

In the same way it is possible for succussion to be produced in a large pulmonary cavity, and I have more than once seen such cavities diagnosed on this account as pneumothorax.

However, succussion in a pulmonary cavity is rare, and chiefly for this reason, if for no other, that the fluid contained in such cavities is too viscid and tenacious to splash easily.

A cavity which contains air alone cannot produce succussion; therefore will succussion not be obtained in a case of simple pneumothorax. When succussion is obtained, it is proof that the cavity in which it is produced contains both air and fluid, and that in the case of the pleura we have not a simple pneumothorax to deal with, but a hydro- or pyo-pneumothorax, as the case may be. Hence it follows that succussion is not obtained in the early stages of pneumothorax, but only at a later period, when effusion has formed; nor is it necessarily obtained even then, if the fluid be in small amount or be very viscid or thick. Usually succussion is easily elicited in hydro-pneumothorax, or where the effusion is sero-purulent; but in pyo-pneumothorax it may be absent, and that even when the amount of fluid is considerable.

Thus, putting aside the rare cases in which a large cavity in the lung or a dilated stomach might cause some difficulty in diagnosis, succussion is practically pathognomonic of pneumothorax with effusion; but the converse is not true, that the absence of succussion shows the absence of fluid.

In most cases the succussion splash has a ringing, metallic character, due, no doubt, to consonance. It is sometimes so loud as to be easily heard at a distance, just as succussion in the stomach is. Patients frequently hear it themselves, and describe it accurately, and may even complain of it.

Metallic Tinkling.—Closely allied to succussion and the bell-sound, and, like them, no doubt, a phenomenon produced by consonance, is the metallic tinkle, a peculiar, musical, ringing sound like that produced by the falling of a drop of water in a grotto (*gutta cadens*). This is indeed the explanation usually given of its production, viz., that a drop falls from the walls of a large cavity into the fluid contained in it, and makes a splashing sound, which, by reverberation from the walls of the cavity, obtains its metallic, ringing character. Similar sounds produced, not in the pleura, but in parts near the pleura, may also in like manner acquire similar characters, *e.g.*, the gurgle of fluid swallowed as it passes down the œsophagus, or the crepitation produced in a cavity in the lung.

"*Bruit de Fistule.*"—Under this name Riegel described a sound not often heard in pneumothorax, viz., a bubbling noise audible on inspiration only, such as would be produced by bubbles of air passing slowly through fluid and breaking on its surface—the explanation which Riegel has given of the phenomenon. The condition under which such sounds could be produced is that in which the opening into the lung is still patent, and the mouth of the fistula below the level of the fluid; so that on inspiration bubbles of air are sucked into the pleura. It is obvious that, for this sound to continue, the fluid must be pressed out of the pleura on expiration, being displaced by the air which has entered on inspiration. If the communication with the bronchus be so free as is obviously necessary, a kind of fluid valve will form, which will be sufficient to prevent the entrance of air, and consequently the occurrence of the phenomenon. In fact, the bruit de fistule is an accidental and altogether rare phenomenon, and is really more likely to be met with in an empyema discharging through the lung than in pneumothorax.

MORBID ANATOMY.—My own statistics are derived from two main sources: first, 101 cases collected from the records of the Chest Hospital, Victoria Park; and secondly, 43 cases from the statistical tables of St. Bartholomew's Hospital; besides which there are 20 other cases which have been under my own personal charge.

Of the 101 cases all but 2 were due to phthisis, and in these 2 phthisis could not be positively excluded. Of these 101 cases, 66 died. The total number of deaths during the period to which the 101 cases belong was 1499, giving an average for pneumothorax of 1 in 23. As about 3 out of every 4 deaths at the Chest Hospital were due to phthisis, it follows that about 1 n every 20 cases of phthisis dies of pneumothorax; in other words, that 5 per cent. is the rate of mortality for pneumothorax in the course of phthisis (57 deaths out of 1175 cases).

The second Brompton Report gives	25 out of	850 cases	—3 per cent.
Dittrich,[1]	14 out of	328 ,,	—4·3 per cent.
Austin Flint, ...	24 out of	700 ,,	—3·5 ,,
Collectively, ...	63 out of	1878 ,,	—3·4 per cent.

Some writers put the percentage higher, but as a rule the numbers then dealt with are smaller, *e.g.*, Douglas Powell (6 out of 60), 10 per cent; Wilson Fox (9 out of 99), 9 per cent; while Habershon,[2] taking fatal cases only, found 114 cases out of 1375 = 8 per cent.

As to the general frequency of pneumothorax among phthisical patients living, there are no statistics upon which any reliance can be placed, nor are such calculations of any practical value.

Biach states that 1 per cent. would represent the average liability of phthisical patients to pneumothorax; but I think it is really much less than this.

Sex.—The sex is specified in 98 cases. Of these 75 were men and 23 women, giving a relative proportion of about 3 to 1.

The St. Bartholomew's Hospital statistics give 34 men to 9 women, or nearly 4 to 1.

[1] *Cf.* Wilson Fox, p. 1104.

[2] *The Hospital*, April 11, 1896.

It is strange that there should be this difference between the sexes; yet most of the statistics show this. Accepting this marked difference as a fact, we must refer it, I suppose, to the greater exposure to strain and effort to which men are subject.

Table of Ages.

	Totals.	—5	—10	—15	—20	—30	—40	—50	—60
Victoria Park Hospital, . .	98	1	1	1	13	55	23	3	1
St. Bartholomew's Hospital, . .	43	2	0	0	4	23	9	3	2
	141	3	1	1	17	78	32	6	3

Age.—Of the 98 cases in the Victoria Park Hospital tables in which the age is given, 78 occurred in persons between 20 and 40, and 13 more between 15 and 20 years of age; so that 91 out of 98 occurred at those periods of life during which phthisis is most frequent and its mortality highest.

The St. Bartholomew's Hospital statistics show the same result, for 32 out of 43 cases occurred between 20 and 40, giving a percentage of 75, while the Victoria Park statistics give a percentage of 77 for the same period.

The youngest case in these series was aged 3 years, and the oldest 60 years.

In the St. Bartholomew's Hospital record no less than 5 cases out of the 43 occurred in children under 5 years of age, and most of these followed tracheotomy. This fact is interesting as showing that in a general hospital it is the rarer and accidental cases of pneumothorax which are more frequently admitted; so that they bear a much higher percentage to the cases due to phthisis than is met with in other places.

Pneumothorax may occur, apart from tracheotomy or traumatism, even in infants.

Thus Ormerod records a case in an infant 6 months old, the result of phthisis. Cases are also recorded in children of 7 months and 11 months by Douglas Powell and Norman Moore respectively. The youngest case known is described by Angel Money at the age of 3 months.

Side affected.—The side affected is specified in 83 out of the 101 cases. It was on the right side in 41, with 28 deaths; and on the left in 42, with 26 deaths. From this it would appear that the liability of the two sides is equal, and that the side affected has no influence upon the prognosis. It is, however, generally stated that pneumothorax is more frequent on the left side than on the right in the proportion of 3 to 2 or 2 to 1.

Taking the combined statistics given in Wilson Fox,[1] the proportion is 5 to 3.

	Left.	Right.	Tolals.
Walshe, ...	55	32	87
Behier, ...	26	17	43
Reynaud, ...	41	27	68
Monuerit and Fleury, . . .	50	25	75
	172	101	273
	63 per cent.	37 per cent.	

[1] Page 1104.

On the other hand, Laennec stated that the right side was most often affected. The question has, however, no practical importance.

The Perforation.—Of the 101 cases 66 died in the Hospital, and there were records of the *post-mortem* examination in 43, in every one of which phthisis was present.

The perforation was found in 25 instances. Weil also found the hole in 26 out of 33, a somewhat larger proportion. The chances of finding the perforation at the time of the autopsy depend chiefly upon the duration of the case; for, the longer the period since the occurrence of pneumothorax, the greater the prospect of the hole becoming closed.

In 19 of these cases the duration is known. It was short in 11, *i.e.*, less than a week, in some only a few hours, in others a few days. In the remainder the duration was much longer; a month in two, six weeks, nine weeks, and eleven weeks in one, and in one as much as four months.

Hence the perforation, though, as would be expected, most easily and commonly found in recent cases, does not necessarily become closed even in those of long standing.

The hole often requires to be carefully searched for, especially when the lung is so completely collapsed as it usually is; but if not found on inspection, it may be necessary, before removing the lung from the body, to cover it with water and then gently drive air into it from the trachea, when, if the perforation exist, the air-bubbles escaping from it will betray its position.

The perforation is, as a rule, single, and this has probably almost always been the case when it is found closed *post-mortem*.

In the 25 cases in which the perforation was still patent it was single in 20; in the remaining 5 there were two holes in 3 cases, and in the other 2, four holes and six holes respectively. Louis gives a case in which there were fifteen perforations.

Of course, in other cases than phthisis, especially where there has been gangrene of the lung, the perforations may be very numerous.

Thus in a case in St. Bartholomew's Hospital Museum, the pleura in the affected part is simply riddled with openings which number thirty or forty; and in a few cases also in which an empyema has burst through the lung and been followed by pneumothorax, the openings may be multiple.

Its size.—In the great majority of cases the hole is small and circular. It is generally one or two lines in diameter.

Walshe mentions, as the largest he has met with, one which was about the size of a fourpenny piece; but I have seen one myself in which there was an irregular opening as large as a five shilling piece.

The oval or circular shape is due to the contraction of the elastic lung round the aperture; for the hole made by a straight incision into the lung with a knife takes the same form. Where there has been a discharge of pus through the lung, the hole may be of larger size; but even then it is, as a rule, under a quarter of an inch in diameter.

The edges of a recent perforation are usually pulpy, soft and caseous; but in cases of longer standing they may be tough and fibrous.

The hole generally leads directly into a superficial cavity in the seat, as a rule, of recent caseous change; that is to say, it is a recent cavity and not a chronic one that usually leads to pneumothorax.

Its position.—The favourite position of the opening is in the postero-lateral region, in the lower part of the upper third of the lung, and therefore usually in the lower part of the upper lobe; but occasionally in the upper part of the

middle or lower lobe. This is, no doubt, to be explained by the fact that in most cases the upper part of the pleural cavity is obliterated by adhesions, and that the perforation takes place immediately below them.

Seat of Perforation.

	Behier.[1]	Chambers.	West.[2]	
Upper lobe, . . .	15	10	10	Apex, 6. Middle, 1. Lower, 1. Lower posterior, 1. Unspecified, 1.
Middle lobe, . . .	7	2	4	Mid-lateral, 2. Anterior, 1. Near sternum, 1.
Lower lobe, . . .	7	5	5	Upper, 1. Upper and anterior, 1. Mid-lateral, 2. Lower lateral, 1.
Upper and lower lobe, .	1	1	0	
	30	18	19	

I do not know that the position of the perforation can be definitely associated with any other fact of importance; but it is interesting to remember that it is in the mid-axillary region that the sudden pain is usually felt when the pneumothorax develops.

When the openings were multiple they were found in the upper third of the upper lobe in 1 case (six openings), and in the lower part of the upper lobe in 1 (two openings), and in both upper and lower lobes in 3 cases. In one of these latter cases there were three openings in the lower part of the upper lobe, and one in the upper part of the lower. In another there were two openings, one in each of the lobes. Besides these, there was another case in which there were two openings, both in the lower lobe, one inch only from the base.

The Amount of Previous Disease.—There is no necessary relation between the amount or extent of phthisical change in the lung and the occurrence of pneumothorax. It may be stated that, as a rule, the liability to the affection is greatest in rapidly-advancing cases, *i.e.*, in those in which the pleural adhesions are either not extensive or have not had time to become firm, and, as a corollary of this, it follows that pneumothorax does not necessarily occur on the most affected side. Indeed, in no inconsiderable number of cases the amount of disease in the lung is quite small; and it may even be that the only focus of disease is the cavity, and that, perhaps, quite a small one, which has ruptured. One or two examples of this will be referred to later, and the fact is of importance because of its bearing upon the question of pneumothorax occurring in apparently healthy persons; that is to say, in those who, previous to the pneumothorax, have presented no obvious signs of disease of the lung.

Wintrich states that pneumothorax may occur in phthisis as the result of rupture of an emphysematous part of the lung near a chronic cavity. I have never myself seen anything *post-mortem* which could support this view, but I hardly venture to deny its accuracy.

In most cases the perforation leads directly into a small cavity, which, on the other side, communicates with a bronchus, so that there is a free passage for the air from the air-tubes into the pleura. Occasionally there is a longer track through the lung before the bronchus is reached. This, however, does not so often occur in the lung itself as outside it through a thickened pleura.

[1] *Cf.* Wilson Fox, p. 1105.

[2] West, *Lancet*, May 3, 1884.

Thus I have seen a sinuous track of 2 inches or more in length, running from the middle of the apex downwards, and opening where the adhesions over the upper lobe ended. In this case there can be no doubt that suppuration had occurred in connection with the cavity, which had tracked downwards and ultimately burst into the pleura.

The orifice is occasionally described as valvular, and accordingly pneumothorax has been described as open, closed, or valvular—open when the perforation is patent, closed when it is closed, and valvular when it permits the entrance of air during inspiration, but not of its exit or expiration. This is a clinical rather than a pathological classification, for there is nothing seen *post-mortem* which resembles a flap or valve. The hole, it is true, may be itself closed during expiration, or, if the hole remain patent, its communication with the bronchus may be occluded. When a cavity perforates the pleura, the walls round are usually soft and flaccid for some distance, and on expiration are forced inwards. In this way the edges of the hole may be brought together; or the cavity, if it be a small one, may be closed completely. This is, I believe, the actual mechanism of the valve action when it is present.

Effusion.—Effusion occurs in most cases of pneumothorax which last any time. A few, it is true, recover completely without effusion, but these form but a small proportion of the total number, while in others death occurs before there has been time for any fluid to develop.

Monneret and Fleury[1] give 16 cases out of 147 in which no fluid formed, *i.e.*, approximately 11 per cent. In 5 of my own cases there was no fluid, the pleura being quite free from all inflammation in 3, and slight dry pleurisy only being present in the other 2. Five cases out of 43 would yield about the same percentage, viz. 11.

The effusion is not necessarily purulent in character, as was formerly taught.

In 20 out of the 43 cases[2] the nature of the effusion was carefully noted; serum was found in 4, sero-pus in 8, and pus in 8. In other words, the effusion was serous or sero-purulent in 12 out of 20, *i.e.*, in 60 per cent., and purulent in 40 per cent. This accords with statistics of Weil and Netter.

Weil,	.	.	26	serous or sero-purulent,	16	purulent;	.	total, 42
Netter,	.	.	13	,, ,,	3	,, .	.	,, 16
			39		19			58

It seems, therefore, that, speaking generally, the chances of the effusion in pneumothorax being serous, sero-purulent, or purulent, are about equal; in other words, that a purulent effusion is met with only in one case out of three. As will be seen, this is a fact of some importance in prognosis.

The serous effusions, as a rule, contain only the tubercle bacillus, and this may be demonstrated sometimes by microscopical examination of the sediment; but, more often, its presence can only be proved by inoculation.

The purulent effusions also contain tubercle bacilli, which are often more easily demonstrated than when the fluid is serous; but other organisms are present as well; for example, the staphylococcus and putrefactive organisms, as well as the encapsulated bacillus of Friedländer and others.

Whatever the organisms are, they must be derived from the cavity in the lung which has been ruptured, and many of these cavities already contain other bacilli than those of tubercle.

The serous effusions often retain their character for a long time, and if they become purulent, the change is due to subsequent infection with pyogenic organisms, *i.e.*, with other organisms than the tubercle bacillus.

[1] Netter, Charcot, *Med.*, Art. Path.

[2] West, *Lancet*, May 3, 1884.

In most cases of long duration the effusions spontaneously become sero-purulent or purulent sooner or later, and this is almost without exception the case when the side has been opened either intentionally or spontaneously.

The effusion is not infrequently purulent from the commencement, and may then rapidly reach a large amount.

Thus in one case 2 pints of pus were found four days after the onset of pneumothorax. On the other hand, even in cases of long duration, the amount of pus may be small. It has been known to reach 2 ounces only after eleven weeks, and 13 ounces only after four months. As a rule, when pus is present, the longer the case lasts the larger the effusion becomes.

Serous effusions indicate less serious irritation, and are often of small size.

Thus in 2 cases of four days' duration, only 2 ounces of serous fluid were found, and in another of a week's duration 3 ounces only.

On the other hand, they may be of rapid development.

Thus, 10, 15, and 20 ounces were found in 3 cases of less than four days' duration, and in some other cases of short duration an even larger amount was found, though not actually measured.

A serous effusion may remain serous for a very long time, and that, too, even if paracentesis has been performed more than once.

I have seen one case of the kind in which the effusion still remained serous two years after the pneumothorax had occurred.

The Air in the Pleura.—The quantity of air in the pleura *post-mortem* may be very large, for the pleura is often distended to the uttermost. The absolute amount is, however, of little importance, for this will vary with the size of the individual thorax.

Its composition.—The air in the pleura varies somewhat in composition in different cases. Thus if it enter from without through a wound in the chest walls, it is, of course, to begin with, simply atmospheric air. If it enter through a perforation in the lung, its composition will be somewhat different, and approach more nearly to that which is found in the vesicles of the lung. This is fairly constant, and has approximately the following percentage composition:—Nitrogen, 79·5; carbonic acid, 4·38; oxygen, 16·0.

If the opening remain patent, the air changes little in composition. If, however, the opening be closed, changes begin at once. The oxygen is rapidly absorbed, and may before long entirely disappear, while the carbonic acid increases considerably, at any rate at first, the nitrogen for a time remaining stationary. Experimentally this has been shown in animals by consecutive analyses of air injected into the pleura for the purpose.[1]

The amount of carbonic acid present in the gas has been used as a test of the patency of the opening.

Thus Ewald[2] has shown that in open pneumothorax, that is to say, where there is a free communication with the lung, the carbonic acid averages about 5 per cent.; and that if the opening be closed completely, it may reach 15 to 20 per cent. He therefore formulated the proposition that if the carbonic acid does not exceed 5 per cent., the opening is patent; if it exceed 10 per cent., it is closed; if it lie between 5 and 10 per cent., the opening is probably valvular. I do not know that the clinical importance of these facts is as great as it might appear.

The earliest analyses of the gas in the pleura were made by Dr. Davy,[3] whose results were—nitrogen, from 88 to 90 per cent.; oxygen, 2·5 to 5·5 per cent.; carbonic acid, 6 to 8 per cent., with traces of H_2S or $(NH_4)_2S$ if putrid fluid were present. These results are merely interesting as a matter of history.

[1] Rodet et Pourrat, *Soc. de Biologie*, 1892.
[2] *Charité Annales*, 1875, vol. ii.
[3] *Philosoph. Trans.*, 1820.

I do not know that any systematic analyses have been made of the gas during the stage of the disappearance of a pneumothorax in man. We know, however, that the oxygen may, in time, entirely disappear (Demarquay [1]), while, for a time, the carbonic acid increases; but as all the gas, even the nitrogen, is ultimately absorbed, it is evident that the composition of the gases must vary greatly at different periods.

COURSE — MORTALITY — DURATION.—As long as dyspnœa continues urgent, the patient is in grave danger; and the greater the danger the more urgent and continued the dyspnœa. If the dyspnœa subside, the risks are of another kind, and vary with the nature and amount of the effusion which forms. If no effusion form, the air may be absorbed and the pneumothorax disappear. Yet, in any case, the patient may die, and that before long, not of the pneumothorax or its consequences, but of the disease which caused it, *i.e.*, of phthisis. Many of these points will be more fully discussed under the heading of Prognosis.

Mortality.—In a disease which is so fatal as pneumothorax, the mortality and duration stand in close relation to one another.

My own statistics are, as stated, based on three series of cases; the first, 101 cases from the records of the Chest Hospital, Victoria Park; the second, a series of 43 cases from the records of St. Bartholomew's Hospital; and a third of 20 cases which have been under my own personal care.

Of the first series of 101 cases, 66 died, giving, roughly speaking, a mortality of 2 out of 3; but of these 101 cases, in 74 the pneumothorax developed in the hospital, of which 57 died, *i.e.*, 77 per cent.; while, of the 27 cases of pneumothorax which were admitted into the hospital with pneumothorax, only 9 died, *i.e.*, 33 per cent. The difference between the two sets of figures is to be explained by the fact that the mortality of pneumothorax is greatest in the first few days which follow the onset; while the latter group of cases consist entirely of those who had survived this period for varied lengths of time. The rate, therefore, given by the first group probably approaches more nearly to the true mortality of the disease than the other.

In the second series of 43 cases, 26 died, giving a rate of 60 per cent., and this is also the rate yielded by the third series of 20 cases under my own care.

We may conclude, therefore, that the general mortality of pneumothorax ranges somewhere about 70 per cent., *i.e.*, rather more than 2 cases out of 3 die.

Saussier's figures give a much higher rate, viz., 131 out of 147, or about 90 per cent.

I think we may fairly claim that pneumothorax is sharing in the general diminished mortality of diseases of the pleura, and this I would attribute to the better comprehension of the true nature of the disease and its dangers, and to the better treatment of the cases during the early days of the attack, when the risk is greatest.

Duration.—In the first series of 101 cases, the duration is known exactly in 39, and approximately in 37 others.

Of the cases in this latter group, the pneumothorax occurred in 10 patients during their stay in the hospital; but the patients recovered sufficiently to leave, so that the terminus *ad quem* is not fixed. In the 27 other cases the patients were admitted with it, and in many of these the terminus *a quo*, *i.e.*, the date of onset, was not quite certain.

Altogether pneumothorax occurred 74 times in patients while they were in the hospital, and in these cases the duration is known in 31 exactly, and very nearly in 8 more; while in the remainder, all that can be said is that the disease did not last less than a certain time, that is to say, the length of the stay in the hospital.

[1] *Gaz. Medic.*, 1865, p. 496.

Table showing Duration in 76 Cases.

Duration.			Duration known. (39 cases.)		Duration Approximate. (37 cases.)			
					Admitted with Pneumothorax.		Developed Pneumothorax in Hospital and Died.	
Months.	Weeks.	Days.			Died.	Discharged.		
1	1	1	10		...	...	4	45 = 60 per cent.
		2	3					
		3	...					
		4	4	18				
		5	...					
		6	1					
		7	...					
				+ 8 = 29				
	2		3	...	...	...	1	
	3		2	= 6	...	...	2	
	4		4		2	...	1	
2	5	...	...		...	...	...	9 = 12 per cent.
	6	...	1		5	8	...	
	7	...	...		...	...	...	
	8	...	...		...	...	...	
	9	...	1		1	1	1	
3	...	...	...		1	7	1	54 = 73 per cent.
4	...	...	1		...	...	...	
5	...	...	1		...	1	...	
9	...	...	...		...	1	...	

Of the 39 *cases in which the duration is known*, 10 patients died on the first day; 2 within twenty minutes and thirty minutes respectively of the attack; the other 8 within a few hours. In all 18 had died by the end of the first week, and 21 by the end of the fortnight; to which may be added 8 more cases, in which the duration cannot be quite determined, though it was certainly less than fourteen days, making thus 29 out of 39 cases which were fatal within the fortnight, *i.e.*, nearly 75 per cent.; in other words, 3 out of every 4 cases.

Six more died within the second fortnight, making thus 35 deaths out of 39 cases within the month, or 90 per cent., a figure which exactly agrees with Saussier's statistics already referred to.

I believe these statistics are too high for the results of the present day, as will be seen when I refer to the more recent series of my own personal cases.

Of the remaining 10 per cent., or whatever the exact figure may be, *i.e.*, of those patients who did not die within the month, the duration was considerably longer, many weeks, occasionally many months, and some patients recovered completely.

Of the 35 *cases in which the duration was uncertain*, 9 were admitted with pneumothorax, and died in the hospital; 10 developed pneumothorax in the hospital, and left at varying dates after the attack, some of whom were known to have died subsequently at their own homes; while 18 patients were admitted with pneumothorax and left with it. Of this second group, taking all the cases together, 10 died within the first month, 8 more within the second, and 2 more within the third, making 19 in all.

Taking the two series together, of 74 cases, 45 died within the month, *i.e.*, 60 per cent.; 9 more died within the second month, making 71 per cent.

Of the remainder, the duration may be much longer, and some may recover; thus 7 were known to be alive at the end of the third month, 1 at the end of the fifth, and 1 at the end of the ninth.

Of the 20 cases which were under my own care, 12 died and 8 recovered, giving a mortality of 60 per cent., which is exactly that which the records of St. Bartholomew's Hospital for the last fourteen years also yield.

The Cause of Death.—The real mortality of pneumothorax is difficult to arrive at; for it often happens that the patient gets over the pneumothorax, possibly even recovers completely from it, and yet dies before long, either of the disease which caused the pneumothorax or of the results to which the pneumothorax indirectly led.

The causes of death must be dealt with in three groups: the first, in which death is the direct result of the pneumothorax itself; the second, in which it is the indirect result of it, and due to the complications which follow, *e.g.*, empyema; the third, where it is the result of the original disease, *e.g.*, phthisis, to which the pneumothorax itself was due, the pnemothorax being at the most but a more or less serious complication, possibly accelerating the end, though not itself entirely responsible for the result.

1. The great majority of the fatal cases of pneumothorax die, as already stated, within the first few days of the disease; nearly one half, 46 per cent., die within the first week.

Of the cases that were fatal during the first fortnight, no less than one-third died on the first day; 2 within twenty minutes and thirty minutes respectively of the onset of the disease, and the majority of the others within a few hours.

Where death is the direct result of pneumothorax, the cause of death is suffocation. This is due in most cases, as already stated, to the sudden embarrassment of the respiration and circulation consequent upon the disease, and it is not so much the amount of change as the suddenness of it that is the cause of the grave symptoms; so that if time be given for the circulation and respiration to adjust themselves, the urgency of the symptoms may pass off, and thus it may be stated, that every hour the patient keeps alive, the better the chances are of his passing safely through the initial dangers.

Another, and different, cause of suffocation, mechanical rather than physiological, lies in the sudden discharge of the contents of cavities into the air-tubes, which get sucked into the remaining lung and thus choke the patient. This I have seen occur more than once.

Lastly, there is a class of case in which death may be called sudden or immediate, and may be referred to shock.

This is very rare, and I have never seen an instance of it myself. Lebert describes a case in a young medical man of the age of 28.

In the 2 cases in which the patients died within twenty minutes and thirty minutes respectively of the onset of the disease, the cause of death was acute suffocation and not shock.

The initial dangers can, no doubt, be greatly diminished by the early recognition of the disease and its appropriate treatment, and it is probable that the diminished mortality of pneumothorax in recent years is due to improved diagnosis and treatment.

2. Where death follows at a later period, it is often the result of the effusion consequent on the pneumothorax.

As already stated, the effusions are, roughly speaking, serous in one-third of the cases, sero-purulent in another third, and purulent in the remaining third.

Where the effusion is purulent, sooner or later the side is opened, either because the empyema points or discharges itself, or because it is deliberately incised. The condition then becomes one of an open empyema, and under unusually unfavourable conditions; for, owing to the prevailing prejudice against early incision in pneumothorax, the lung may have become bound down and cannot subsequently expand; and secondly, because of the complication of the effusion, with more or less extensive tubercular disease of the lung. Any way, the patients die usually of asthenia, the result, partly, of the prolonged discharge, and partly of the phthisis.

3. In the third group of cases it is not of pneumothorax that the patient dies, but of the original disease which produced the pneumothorax. This is, in most cases, phthisis.

In many of these cases the pneumothorax is nothing more than a more or less grave complication, which may accelerate the end, though it be not of itself the cause of death; in other cases, though the patient is actually dying of phthisis, the pneumothorax which has been produced may get completely well.

PROGNOSIS.—In dealing with prognosis, we must presume that we have a case of complete and not of partial pneumothorax to deal with, for localised pneumothorax is comparatively an unimportant affection; also that when pneumothorax results from the discharge of an empyema through the lung or externally through the chest walls, the prognosis is that of empyema and not of pneumothorax, and therefore relatively more favourable.

The three considerations usually embraced by the common term 'prognosis' require, in a case of pneumothorax, to be separately dealt with. They are, first, the immediate risk to life; secondly, the possible duration of life; and, thirdly, the chance of ultimate recovery. Many of these points have been already incidentally referred to in treating of the mortality and duration of the disease.

I. *As regards the immediate risk to life.*—This is always great, and greatest during the early hours and days of the disease, as has been already shown; so that, speaking generally, other things being equal, the chances of life increase in proportion to the period which elapses from onset; but in any given case the actual prognosis at the time must be determined by many other considerations, of which the following are the most important:—

(1) The urgency of the symptoms.
(2) The effect produced upon the opposite lung, and the amount of previous disease in it.
(3) The effect upon the heart.
(4) The general strength of the patient.
(5) The nature of the cause, *e.g.*, injury, phthisis, gangrene of the lung, etc.

(1) *The urgency of the symptoms.*—This is measured practically by the amount of dyspnœa and cyanosis, which is often so extreme that, unless relief can be given, the patient must die. The greater the relief given by paracentesis, and the longer the interval between successive paracenteses, the better the immediate prognosis.

(2) *The condition of the opposite lung.*—This lung, being overworked, may not be equal to the strain upon it, and if it give way, the physical signs of congestion (*i.e.*, of bronchitis) will manifest themselves. Thus it is that rhonchus and sibilus become signs of very bad omen.

The amount of previous disease in the lung is important, inasmuch as the lung is rendered thereby less able to meet the extra demands made upon it.

(3) *The effect upon the heart.*—The difficulty of circulation throws extra work upon the right side of the heart, and if this exceed a certain amount, dilatation and its consequences will be the result, a result which will be the more likely to follow if the heart be previously diseased or enfeebled.

(4) *The general strength of the patient.*—This is important in two respects—first, in so far as it affects the muscular power; for this is taxed, on the one hand, in the respiratory muscles which have so much extra work to do, and on the other, in the heart, which, in a weakly patient, is all the more likely to give way rapidly: secondly, in so far as it affects the general stamina or powers of endurance of the patient.

(5) *The nature of the cause.*—Lastly, a good deal will turn upon the disease which has led to the pneumothorax. In the vast majority of cases this is phthisis, and this class of case may be taken to represent the average.

As giving a prognosis better than the average, may be instanced cases of pneumothorax following simple injury, such as a stab or a fractured rib, and as giving a worse prognosis, cases of gangrene of the lung, for these are almost invariably fatal.

II. *As regards the duration of life*, if the urgent initial symptoms have passed off, the prognosis will depend upon the general strength of the patient, and upon the nature of the disease with which the pneumothorax is associated; but principally upon the complications to which the pneumothorax may lead, *i.e.*, upon the occurrence of effusion and its character.

III. *As regards recovery.*—Speaking generally, the number of recoveries in pneumothorax is small. Most statistics give a more or less complete recovery in not more than 10 per cent.

The figures derived from the St. Bartholomew's records yield over 25 per cent., and my own cases are even more favourable still.

In my own 20 cases, 5 recovered completely and remained well for long periods; in 3 the recovery from pneumothorax was complete, but the patient died subsequently of phthisis; and in 3 others recovery was incomplete. Besides these, I have seen one other case in consultation in which recovery was complete, and the patient has remained well for many years.

The most favourable cases are those in which no effusion takes place. The air is then simply absorbed, and often with considerable rapidity. Of this result there were 4 instances, and to these may be added the consultation case referred to.

In another group of cases effusion forms, but it is serous, and recovery then takes place, with or without paracentesis, just as it does after ordinary serous effusion.

Sometimes the fluid, after being removed by paracentesis, does not re-form; but the air remains for some time longer, though it is ultimately absorbed also. In other cases the air is absorbed first, and fluid takes its place; the case then becomes one of ordinary serous effusion, and recovers in the same way.

When the effusion is purulent, the prognosis is much more serious; but even some of these cases, in time, recover. In one of my cases expectoration took place through the lung, and in the end the patient recovered completely. In one other the pus was discharged externally and recovery was imperfect, the patient being left with a discharging sinus for more than twelve months, by which time extensive phthisis had developed, of which the patient before long died.

CASES OF RECOVERY.

1. Without Effusion of Fluid (Simple Pneumothorax).

CASE 1.—*Acute phthisis—Pneumothorax of the left side developing in the hospital—Complete recovery in five weeks—Rapid progress in the phthisis, and death subsequently, at home, from phthisis.*—Martha L., aged 19, was admitted into the Chest Hospital, Victoria Park, with acute phthisis of the right apex, and with some early mischief on the left side also. The disease was probably of about three months' duration. On April 7, six weeks after her admission, she was seized one morning with pain in the left side and some shortness of breath. The symptoms were not very urgent, but she was found, on examination, to have pneumothorax of the left side, with considerable displacement of organs. No fluid formed; the air was absorbed, though somewhat slowly, and by May 14th (*i.e.*, five weeks from the onset) the two layers of the pleura were in contact in most places and dry friction was heard, and by the end of the month (*i.e.*, seven weeks from the onset) the condition of the side was the same as before the pneumothorax; the heart's apex was in its normal place, and over it a well-marked pleuro-pericardial friction was present, which persisted for a short time and then also disappeared.

The lung re-expanded completely, and nothing was left but a little impairment of percussion at the base behind. There had been no evidence at any time of fluid, and succussion had been entirely absent.

The temperature was hectic throughout, and was completely unaffected by the occurrence of pneumothorax.

On July 10th she left the hospital at her friends' request, *i.e.*, about twelve weeks after the onset of the pneumothorax. The phthisis had all the time been making very rapid progress, and a few weeks later caused the patient's death.

CASE 2.—*Pneumothorax, left side, without definite symptoms at time of onset, without effusion, and with complete recovery.*—Samuel D., aged 46, a miner, was sent up to me by Dr. Parry Jones, of Pinxton. About six weeks before admission he was at work, and felt a pain in the left side, which made his breath somewhat short. The dyspnœa was not severe, so that he was able to continue his work, and he remained at work until shortly before I saw him, with no special discomfort except that he found his breath a little shorter than usual on effort. For this he consulted Dr. Parry Jones, who found that the patient had pneumothorax, and sent him up to the hospital to me, although the man did not think himself ill enough to be placed upon the sick list.

The whole of the left side was tympanitic to percussion almost to the very margin of the ribs; the heart was displaced, so that impulse was felt below the right nipple, and the pleura extended an inch and a half to the right of the sternum, opposite the third rib. There was no bell sound or succussion; the breath sounds were absent everywhere except in the interscapular space and a little below this, and here the breathing was exaggerated and some metallic tinkling heard. The movements of the side were quite free.

I made the following note at the time:—The patient has pneumothorax of the left side. The amphoric breathing and metallic tinkling are probably produced in the lung, but the opening itself is closed. The free movements of the side indicate that the lung is not bound down, but expands readily on inspiration.

On 3rd September the patient was quite well, the air completely absorbed, and the lung re-expanded. The following note was then taken:—The percussion is equal on the two sides, the cardiac dulness in its normal place; the stomach resonance is a little higher than normal; over a small spot, at the base behind, there was probably a little pleurisy, for the percussion was slightly impaired, the breath sounds feeble, and some fine crepitation, probably pleuritic friction, heard on deep inspiration. Except for this, the voice- and breath-sounds were normal over the whole side. The patient gained 14 lbs. in weight in the hospital, and looked and felt quite well.

Once or twice during his stay he had a little hæmoptysis, from which he had suffered occasionally before his attack.

Dr. Parry Jones was kind enough to send me the following report of him, ten years after his attack of pneumothorax:—"I find Samuel G. has been at work ever since 1886, and has had no illness. He appears to be now in good health, and his chest is in every way normal. I hear, however, from a doctor who saw him a few weeks ago, that he had then a very slight attack of pleurisy on the left side, but of this there is no sign left. He appears to be very vigorous, and has recently figured in an affiliation case."

CASE 3.—*Man, under treatment for phthisis, developed right pneumothorax—Complete recovery in six weeks—Well twelve months afterwards.*—Alfred B., aged 24, had been under observation for some time, suffering from phthisis, but without any very marked physical signs. On 7th November he was suddenly seized with severe pain on the right side, and dyspnœa, and

was taken to the Royal Chest Hospital, where Dr. Hensley drew off a large quantity of air next day by means of the aspirator. The patient left that hospital and came under my care four days later. The dyspnœa had disappeared, but the physical signs of pneumothorax were obvious. There was no distension of the side to sight or touch, but there was a good deal of subcutaneous emphysema which had followed the paracentesis.

The heart was displaced, so that the apex beat was felt 1 inch outside the left nipple line; there were loud bell sounds and amphoric breathing over the whole side; succussion was absent. On 15th November the physical signs were the same, the bell sounds and amphoric breathing well marked. On 20th November the physical signs were unaltered, but the apex of the heart had returned to the nipple line. On 9th December the bell sounds had disappeared, and did not subsequently return; the amphoric sounds were very weak, and the heart's apex was within the nipple line. On 20th December the right side seemed to be a little flatter and to move somewhat less than the left, but otherwise to be normal; the amphoric breathing had also disappeared, and natural breathing was heard over the whole side, back, and front, while the apex of the heart was in its normal place.

Thus within six weeks the air had been completely absorbed and the lung had re-expanded; the patient seemed perfectly well and was discharged. He was often seen subsequently, and, though troubled from time to time with cough, remained in better health than usual, and, indeed, gained flesh.

With the exception of a little streaky hæmoptysis five months after his discharge, he remained perfectly well, and was known to be in good health more than a year after the occurrence of the pneumothorax.

Case 4.—*Pneumothorax, right side, probably third attack, no fluid—Complete recovery.*—William D., aged 22, was delicate as a boy and was subject to cough, but of recent years had grown stronger, and become fairly active and strong. He was in his usual health until last August, when, whilst walking, he had a sudden attack of pain just above the right clavicle, and felt a bubbling sensation in the right side, and the breath suddenly became short. After this he noticed that he had a "churchyard cough," but he was able to keep to his work, but obliged to avoid all violent exercise for about a fortnight, when he got quite well.

In the beginning of November, that is to say, four months later, after a game of football, in which he was, on more than one occasion, heavily charged, he became again short of breath. This lasted about a month, but in the end he again got perfectly well. He remained well until just before Christmas (23rd December), when suddenly, after getting up in the morning, the shortness of breath again appeared, and continued so until his admission into the hospital on 4th January. He was then found to have pneumothorax of the right side. The chest was fairly well developed, but was a little flat in the upper part; the heart's apex was in the left nipple line, but the pleuritic hyper-resonance extended across the sternum for nearly an inch to the left. On the right side the breathing was amphoric in character, but no bell sound was obtained and no succussion. The liver dulness in front was at the lower edge of the seventh rib, so that the tympanitic percussion did not extend exactly to the costal arch.

The diagnosis made was that the patient had had pneumothorax, and that the air had been partly absorbed. The patient presented no symptoms; the temperature was normal and he seemed well, so that on the 17th he was allowed to get up, and on the 24th was up all day. He could get about without any shortness of breath, and the physical signs were not much altered.

On 6th February the following note was taken:—Shape of the chest symmetrical; movements free, but a little more expansion on the left side than the right on deep inspiration; liver now stands at the sixth rib in the nipple line on the right side; cardiac dulness normal in place and size. Percussion note still a little hyper-resonant on the right side, the left normal.

The breath sounds vesicular and audible down to the base, though a little more distinct at the apex than elsewhere. A little fine crepitation in the mid-axillary line was heard, where some pain had been complained of.

The patient was seen many times subsequently, and was known to be in excellent health for many months, when he became lost to observation.

Case 5.—*Pneumothorax in an apparently healthy girl—Recovery.*—The following account of the case was sent me by Dr. Waldo of Clifton:—"The young lady, aged 15, thought she felt something give way in her right side when dancing. She was first seen by me on 14th August 1890, when she complained of a sharp pain under the right collar bone. The heart's apex was half an inch outside the nipple line on the left side, and there were the ordinary signs of pneumothorax, but no succussion. Shortly afterwards you saw her and recommended that she should stay in bed for some weeks. I have a note that on the 6th of October 1890 the heart's apex was in its normal position, and the respiratory murmur equally good on each side. The patient had never spat any blood, and there were no signs of tubercle. Her weight was 6 stone 14 lbs. I have seen the patient only a few times since, as far as I can recollect, and I have every reason to think that the patient is still in good health."

2. With Serous or Sero-purulent Effusion (Hydro-pneumothorax).

Case 6.—*Pneumothorax with sero-purulent effusion of three months' duration.*—William G. P., 22, a labourer, was quite well till three months ago, when he was attacked at night with sudden dyspnœa, so that he fainted. He got gradually worse, had some cough and expectoration, spat a little blood a few times, but only about an ounce at the most; began to sweat at night and to lose flesh. After a few days he got better, but was never well enough to go out. He remained in the same condition till a pain in the right side set in, which gave him a great deal of distress, so that after a few days' suffering he applied for admission to the hospital and was taken in, three months after the original attack. He was then found to have hydro-pneumothorax of the right side; the heart's apex was an inch and a half outside the left nipple; as he lay in bed the whole of the front of the right side was resonant down to the margin of the ribs; the liver was displaced in the usual way; the breath sounds were completely absent; the bell sound was well marked, and so was succussion, which had a metallic character.

There was a considerable amount of fluid in the chest, the level rising, on sitting forward, up to the sixth rib in the nipple line. The temperature was slightly raised, reaching 100° or 101° every evening, but dropping to 99° at other times of the day.

The patient was admitted on 4th May, and by the 22nd of May the side had become much more distended.

The superficial veins on the right side of the thorax were greatly dilated, the course of the blood being from above downwards; so were also the veins of the right arm and the right side of the neck. It was evident that there was considerable intrapleural pressure, although but little dyspnœa was experienced when the patient was lying still. The displacement of organs was extreme.

On the 25th May the patient was aspirated. Air was allowed to escape as long as it would.

The gas was analysed roughly, and appeared to contain about 12 per cent. of carbonic acid. There were removed by the aspirator 41 ounces of a sero-purulent fluid with thick flakes, which was faintly alkaline, had a specific gravity of 1020, and became almost solid on boiling. The whole of the effusion could not be removed, as the patient began to cough and feel faint. The heart's apex returned, after the operation, nearly 3 inches in linear measurement, and beat nearly under the nipple.

The intra-pleural pressure was estimated and found to be, on expiration, $13\frac{1}{4}$ inches of water, on inspiration $6\frac{7}{8}$ inches of water, so that there was a respiratory oscillation of nearly $6\frac{1}{2}$ inches.

On examining the patient four days later, it was found that no succussion could be obtained, and that the bell sound was audible only over the upper part of the lung posteriorly, as far down as the spine of the scapula; in the inter-scapular space some distant amphoric breathing was heard, but elsewhere, as before, no breath- or voice-sounds were to be detected.

In the course of the next few days the fluid evidently was accumulating again, succussion, which was absent, returned, and the veins on the chest became more dilated, much as they had been before the paracentesis.

On 16th June a second paracentesis was performed. The pleural pressure amounted to 8 inches of water with a respiratory variation of not more than $1\frac{1}{2}$ inch. No air escaped, but fluid came at once, and 22 ounces of sero-purulent fluid were removed, which, however, contained much less pus than on the previous occasion.

On 22nd June the patient felt and looked better, and breathing was greatly relieved. Succussion was absent. The amphoric breathing and bell sound were heard only about the upper part of the right interscapular space.

By 29th June the apex of the heart had returned almost to its normal place. The bell sound was heard only in the supra-spinous fossa and in the upper part of the interscapular space. There was no succussion. The breath sounds were hardly different in character from those on the opposite side.

Although there was a little dulness on sitting up, it was evident that the fluid had almost entirely disappeared, that the lung had expanded partially in its lower part, and that fluid could not be present to anything more than a slight amount.

One month later the following notes were taken:—"The right side is a little contracted, but the movements are free, especially in the lower parts, though not quite so free as on the opposite side. Percussion is somewhat impaired over the whole side, except at the apex, back and front, where it is still somewhat hyper-resonant; over this area the vocal vibrations, vocal resonance, and breath sounds are less than in other parts, and the bell sound is still audible. Elsewhere the voice- and breath-sounds are fairly normal. The heart's apex is hardly half an inch outside its normal place. The veins of the right arm, neck, and chest are still somewhat dilated." The conclusion was drawn that the pneumothorax had become limited to a very small area at the apex; and although the percussion was a little impaired in the lower part of the chest, that the lung had expanded freely, and was functionating normally.

On 24th August recovery seemed to be almost complete, except that the side was a little flattened, and its movements slightly restricted as compared with the other. The voice- and breath-sounds, though somewhat rougher than on the left side, were otherwise not abnormal ; the apex of the heart was in its normal place, the superficial veins were still a little dilated. The breathing was not in the least short, even on going up and down stairs ; the patient had gained flesh, and, in fact, seemed to be perfectly well.

This case was under observation in the year 1883. I saw the patient last in 1899. The chest appeared absolutely healthy, and it was quite impossible to tell that there had ever been anything the matter with it.

CASE 7.—*Case of phthisis under tuberculin treatment, left pneumothorax—Serous effusion—Paracentesis—Recovery—Recurrence three weeks later, without effusion—Recovery.*—Edward M., 24, seaman, had suffered from phthisis for about nine months, and came into the hospital for the purpose of having Koch's treatment applied. He presented well-marked signs of phthisis at the left apex. The disease did not appear to be active, for the temperature was normal on admission, and though the injections of tuberculin were followed by the usual rise, the temperature soon returned to normal and remained so.

On May 13th the patient suddenly woke up in the night with a violent pain in the left side of his chest, but it extended, he said, from the left ear down to the margin of the ribs. The patient had a rigor, and the temperature rose to 102, the dyspnœa was considerable, so that the patient could hardly speak a word. Pneumothorax was found on the left side, and on 14th May the following note was taken :—

The whole left side was tympanitic to percussion and the heart displaced far on to the right side, and over the whole of it the bell sound was heard. At the base there was dulness, which, in the recumbent position, rose high into the axilla. Succussion was easily elicited. It was evident that besides the pneumothorax there was a large effusion, and on account of its rapid formation its nature was probably serous.

Paracentesis was performed ; a quantity of air escaped, and 22 ozs. of serum were drawn off. The specific gravity was 1018. The intra-pleural pressure on inspiration was +4 inches of water, and on expiration +5, with a respiratory oscillation of 1 inch.

The patient stood the operation well, but towards the end of it complained of pain in the præcordial region, and on auscultation here pleuro-pericardial friction was heard. The heart returned nearly to its normal position.

On 1st June, nearly a fortnight later, the following note was taken :—

"The heart's apex is now only about an inch from its normal position ; the stomach resonance is distinct ; the voice- and breath-sounds are heard everywhere, even down to the base behind, so that there can be but little fluid left. There is no succussion, and the bell sound is gone. It is evident that the air has completely disappeared and the fluid nearly so."

On 8th June the patient, after a violent attack of coughing, was seized again with pain in his left side.

His breath became short, and his temperature, which had been for some days normal, rose again to about 102° ; the bell sound returned in the middle part of the chest ; that is to say, it was not obtained over the apex in front, nor at the base behind.

On 25th June the following note was taken :—The diagram shows the irregular area over which the bell sound is heard. There is no succussion. From the physical signs it is evident that the impaired percussion behind is due to thickened pleura, and that the irregularity of the area in which the air is contained is due to the irregular adhesion of a partly expanded lung.

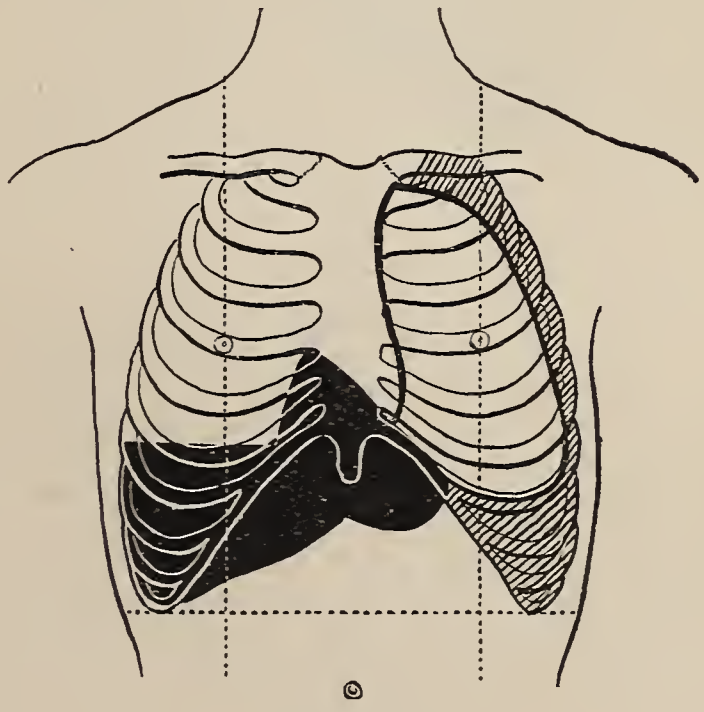


Fig. 160.

Diagram showing the irregular area over which the bell sound was heard.

Gradually the air was absorbed and the lung expanded as before, so that by 10th July the condition was very much that which the patient presented at the time of his admission into the hospital, with the exception that there was a little dulness at the base due to thickened pleura.

On July 24th the patient was discharged from the hospital in much the same condition as he had been on his entry.

Case 8.—*Pneumothorax of the left side in an apparently healthy person without marked symptoms—Considerable effusion, spontaneously absorbed—Complete recovery—Six months later pneumothorax of the right side with much dyspnœa, without effusion, also with complete recovery.*—Arthur F., 21, a well-developed, healthy-looking man, was seized on 6th November with sharp pain between his shoulders.

Later in the day he complained of tightness in his chest and pain on breathing. These symptoms continued, so that he felt obliged to remain at home, though not in bed. He did, during the succeeding fortnight, actually go to work on two days in the week. The pain was worse at night, when he often felt hot, and sweated, and sleep was broken. He had lost flesh and strength rapidly, and feeling ill, came to the hospital. With the exception of a little rheumatism now and then, he had been in perfectly good health all his life, but a few days before the onset of the present illness he had got very wet, and worked all day afterwards in his damp clothes.

It transpired subsequently that on one occasion, 1½ years ago, the patient had once spat up a little blood, about two teaspoonfuls only.

The patient came under observation eighteen days after the onset of his illness, viz., on 23rd November. He was then found to have pneumothorax of the left side.

The whole front of the side was tympanitic, with well-marked bell sound and succussion. A good deal of fluid was present, the level reaching up to the mid-axilla as the patient lay upon his back. The vocal vibration, vocal resonance, and breath sounds were almost absent over the whole side, but had a somewhat amphoric character on coughing.

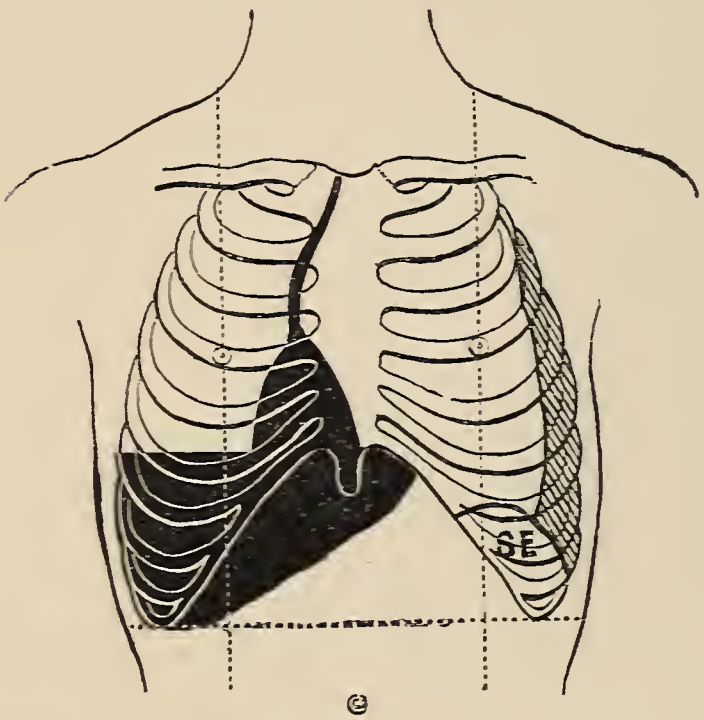


Fig. 161.

Diagram showing the position of the organs. The shading in the axilla shows the level of the fluid. Over the area of stomach resonance the percussion, though impaired, is not dull, due, no doubt, to some distension of the stomach below.

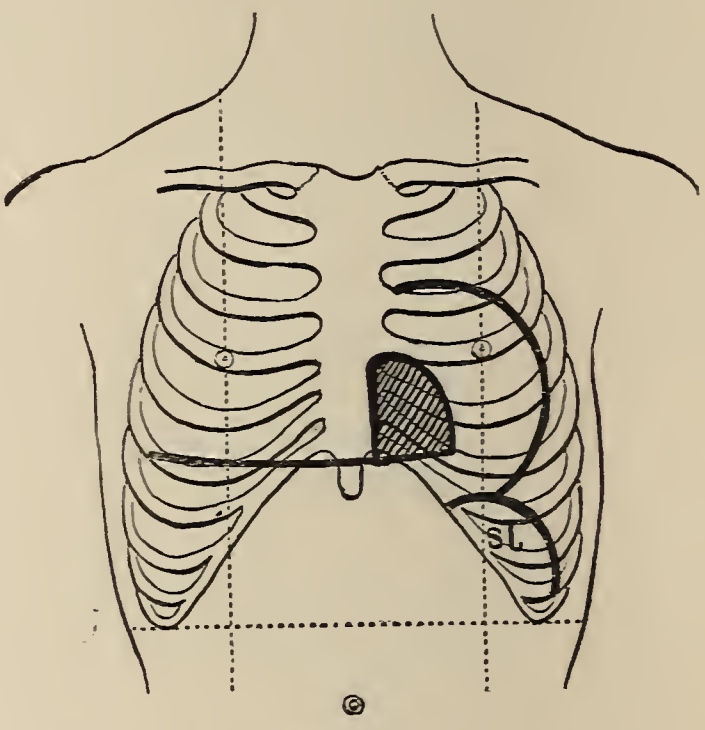


Fig. 162.

Diagram showing the area of hyper-resonance described.

The temperature was 102° at its maximum, but for the greater part of the day a good deal below that. A needle was inserted and clear serous fluid obtained.

As there were no urgent symptoms, the patient was not interfered with; was kept in bed quietly and treated on general lines. By 5th December it was evident that the fluid was being absorbed, and that there was great improvement taking place in the side, and on the 12th the following note was taken (*cf.* diagram):—"Outside, but following the line of cardiac dulness, which is in its normal place, is an area of hyper-resonance about 2 inches in width. Elsewhere the percussion is somewhat impaired, but not anywhere dull. The vocal vibrations are heard and felt over the whole side, though not quite as distinctly on the right, but the breath sounds are hardly audible except in the upper part, and there there is a little crepitation, probably friction, on deep respiration. There is now no longer any bell sound or succussion. The fluid has been almost entirely reabsorbed; the lung has expanded to a great extent above, though it is possible that in the hyper-resonant area a small amount of air still remains in the pleura; at any rate, over this area the bell sound was obtained a few days ago, though it is absent now."

In a few days more even this hyper-resonant area disappeared, and the physical signs became practically normal over the whole side.

On 19th January the patient was discharged, in all respects well, and except that the left side of the chest moved a little less freely than the right, there was nothing to show that the chest had been affected.

About five months later, 15th June, the patient, having in the interval been in good health, was seized in the night suddenly with severe dyspnœa and pain in the right side. He was admitted the next day, and found to have pneumothorax of the right side (*cf.* diagram); the dyspnœa, though urgent, was not extreme. General measures were adopted, with relief, and on 23rd June it was obvious that the air was being absorbed, for the heart's apex was 1 inch nearer the sternum. On the 27th the liver had risen, so that its upper border was at the sixth rib in the nipple line. On the 30th succussion was obtained, though only with difficulty, but there were no other signs of fluid. On 4th July succussion could no longer be obtained. On 7th August the lung was in contact with the chest walls everywhere, and some friction was heard at the base behind. Recovery was now complete.

I have seen the patient lately, *i.e.*, 8 years from his last attack, and he is perfectly well, and the chest shows no signs of abnormality.

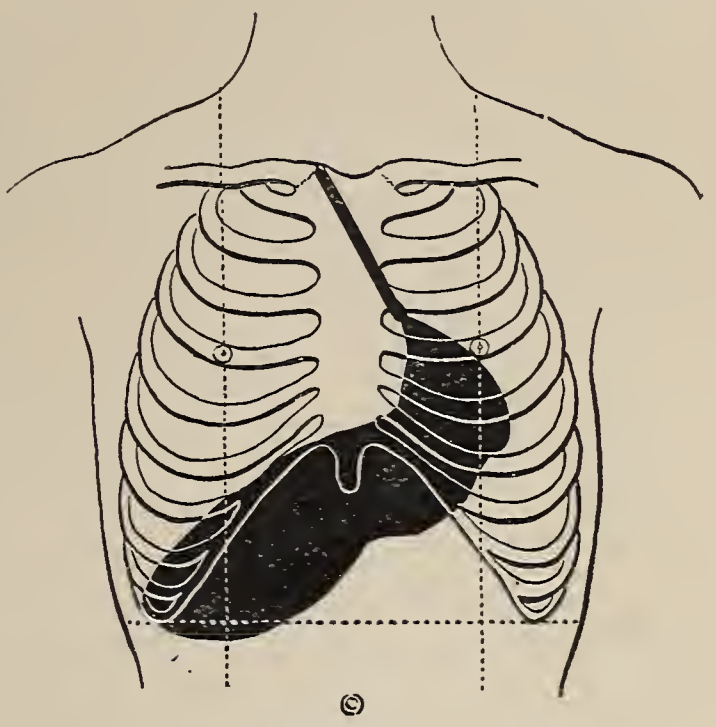


Fig. 163.

Diagram showing the position of organs in the second attack of pneumothorax.

Case 9.—*Pneumothorax with serous effusion—Paracentesis—Absorption of air—Partial recovery.*—Frederick L., aged 31, was in good health, except for a slight cough during the last six months, till eleven weeks ago, when on rising one morning he found that he could not draw his breath freely, and that he had a little pain in his right side. He tried to go on with his work, but found that he was unable to do so, and had to take to bed, where he remained for a fortnight.

After six weeks he was able to go back and do light work up to the time of his admission, though for the last three or four weeks he had found his breathing getting gradually more and more short.

On admission he was found to have pneumothorax of the right side, with a considerable amount of effusion. Succussion and bell sounds were well marked, and there was well-marked ægophony at the angle of the scapula. There was the usual displacement of the heart and liver. The fluid in the recumbent position reached up to the level of the fifth rib, and on sitting up as high as the third rib. The temperature rose to 100° or 101° for three or four days after admission, and subsequently fell to normal.

Three weeks later paracentesis was performed, and 80 ozs. of clear serum removed, with great relief. The intra-pleural pressure was zero. The organs returned nearly to their normal places; the patient was quite comfortable, and suffered no distress from the operation.

Gradually, however, the fluid reaccumulated, and when the patient decided to leave the hospital, a month later, his condition was much the same as on admission. After the patient had been out of the hospital for a month he returned to show himself. The lines of dulness were much as before, but there was no succussion, and the breath sounds were audible all over the resonant upper part. The lower part of the chest contained a good deal of fluid, but no succussion could be elicited. The patient said that he had heard the splashing sound in his right side on leaving the hospital, but that it gradually decreased, and had been absent for the last three weeks.

The patient now passed from observation, and his subsequent career is not known. The air had been all absorbed, and the case had become one of simple pleuritic effusion. However, as he felt well enough to do some work, and would not submit to operation, nothing further could be done.

3. With Purulent Effusion (Pyo-pneumothorax).

Case 10.—*Left pneumothorax—Serous effusion—Paracentesis—Suppuration of puncture—Empyema—Sinus left—Death from phthisis some months later.*—Edward J., 46, had been ailing for twelve months with indefinite symptoms, when he was seized suddenly one night with very severe dyspnœa, with cough and pain in the left side. A fortnight later he was admitted into

the hospital. He was suffering very considerable dyspnœa, was cyanosed, and bathed in sweat, but after being in the hospital for a few hours he rallied. He was found to have pneumothorax of the left side, with a large amount of fluid.

Ten days after admission he was tapped, and 74 ozs. of clear serous fluid were removed. The intra-pleural pressure was 9 inches of water.

After the operation subcutaneous emphysema developed. Fourteen days later he was tapped a second time; the pressure then was $4\frac{1}{2}$ inches on expiration, 1 inch on inspiration, with a respiratory oscillation of $3\frac{1}{2}$ inches. This time no fluid could be obtained.

A short time afterwards the puncture suppurated, and ultimately a communication was formed with the pleura, through which a discharge of thin sero-purulent fluid took place.

From this time the patient slowly improved, the lung re-expanded, and, after four months, the patient left the hospital in a fairly good state of health, with a discharging sinus in his left side. The lung had expanded fairly well over the whole side, but there were obvious signs of phthisis at the left apex.

Some three or four months later he was readmitted into the hospital because his health had been failing. The condition in the side was much as it was before; the lung had expanded over the greater part of the chest, and the empyema cavity was quite a small one. As the discharge did not seem to be quite free, the opening was somewhat extended with the knife and a drainage tube put in. The phthisis made rapid strides, and a few months later, about twelve months from the commencement of the pneumothorax, had involved both sides. Ultimately the patient died of phthisis.

Case 11.—*Left pyo-pneumothorax—Discharge through the lung—Partial recovery.*—Walter C., 29, came under observation with left pyo-pneumothorax. No definite history of its onset could be obtained. The patient had been ailing for three years with a cough, which had been much worse during the last twelve months. He had occasionally spat a little blood, and suffered from night sweats and loss of flesh recently.

The signs of pneumothorax were obvious, but succussion was not easy to obtain, and the amount of fluid was not large. The left lung was adherent at the apex, where there were the signs of early phthisis. The symptoms were not in any way urgent, and did not call for any active treatment. In a fortnight both the air and the fluid were reduced in amount, and the patient had gained 3 lbs. in weight. Succussion, also, was more difficult to elicit.

The patient then began to spit up pus, and this soon increased in amount. With the free expectoration of the pus the condition of the patient improved.

Nothing further was done, and in six weeks' time the patient left the hospital greatly improved in general health. The signs of pyo-pneumothorax still persisted, though not quite to the same degree, and some ounces of pus were being expectorated daily. It would, I think, have been well to have operated on this case and laid the side freely open, but this was prevented by the patient's leaving the hospital.

FORMS OF PNEUMOTHORAX.

Partial and Complete.—In partial pneumothorax the affection is limited or localised to a part of the pleura, and that often a small part. It is not a common affection, and occurs most frequently as the result of a localised empyema, when air gains access to it, or where decomposition occurs in its contents. The limitation of the pneumothorax is due to the lung being fixed by adhesions all round it. Some authors maintain that the division is of no value, because, in nearly every case of pneumothorax, there are adhesions more or less extensive at the apex, so that actually the whole pleura is but rarely affected.

What is generally meant, when the term "pneumothorax" is used in its ordinary acceptation, is that the air occupies the whole or very nearly the whole pleura, and clinically there is all the difference between those forms of pneumothorax in which the heart and mediastinum are displaced, and the opposite lung so profoundly affected, and a limited or partial pneumothorax, in which these displacements are absent.

Open, closed, and valvular.—Except where there is a very large opening in the chest wall, so that the air passes out on expiration as freely as it enters on inspiration, all pneumothorax is at first valvular, at any rate more or less, *i.e.*, the air finds more or less difficulty on expiration. Thus the pleura becomes more and more full of air and the lungs more and more compressed, and this

obviously tends to close the hole more or less completely. When the hole is of an ordinary size, it will become patent on inspiration, and thus admit air, but only so long as the pressure in the pleura is less than that of the air in the air-tubes. As soon as the pressure on the two sides is equal, no more air can enter, and the hole remains closed. If the edges cohere, the hole will become permanently closed; if not, so soon as the pressure in the pleura is diminished, as it may be by paracentesis, the orifice may open again and air pass again into the pleura. This is the explanation in many cases of the return of dyspnœa after paracentesis.

The subject has already been referred to more than once, incidentally, but as I do not think the classification has the great importance once attributed to it, it need not be further discussed.

The forms of pneumothorax which deserve further consideration are four—

1. That in which it develops without any special symptoms (*Latent pneumothorax*).
2. That in which it occurs in persons who have, till then, presented no obvious signs of disease (*Pneumothorax in the apparently healthy*).
3. That in which there is more than one attack on the same side (*Recurrent pneumothorax*).
4. That in which it occurs on both sides (*a*) either at the same time, or (*b*) after an interval (*Double pneumothorax*).

Latent Pneumothorax.—This is the name given to those cases of pneumothorax, rare but remarkable, in which the severe symptoms which usually mark the onset are entirely absent, or so slight as not to suggest what has occurred. The cases fall into two groups: first, that in which the patients are already under observation for some grave affection of the lung, usually phthisis; secondly, that in which the patients are apparently healthy at the time of onset.

(*a*) Where the patients have some previous lung disease.

Case 12.—Alice R., aged 21, was under my own care with acute phthisis, the apices of both lungs being affected, the right most. She had been lying entirely in bed for some weeks, and was apparently improving. The usual weekly examination of the chest had been made carefully, and on the day following, the apex of the heart, which had been previously in its normal place, was found beating in the middle of the left axilla. Further examination showed the presence of pneumothorax on the right side. Careful enquiry was made of the patient and of the night-nurse, but failed to elicit the history of any symptom or change observed by the nurse, or felt by the patient during the night when the pneumothorax must have occurred. Subsequently purulent effusion developed, and 80 ozs. of pus were removed by paracentesis. In the end the patient gradually sank and died of exhaustion.

Case 13.—A similar case was under my care in a lad of 16, who was suffering from acute phthisis. The pneumothorax developed on the second day after the patient came under observation; this lad also soon after died of phthisis. The pneumothorax occurred on the side least affected with tubercle; *post-mortem*, 6 ozs. of pus were found in the pleura, and the perforation was patent.

(*b*) Where the patients present no signs of previous lung disease, or where they have been previously in apparently good health.

Case 14.—The first case of this kind I saw occurred in an elderly man, of about 50 years of age, who was admitted into St. Bartholomew's Hospital because he looked ill. His health had been recently failing, and he was thought to be suffering possibly from malignant disease. This patient can hardly be spoken of as previously healthy, but, at any rate, he presented no definite signs of disease, and owing to the absence of urgent symptoms, and to the admission at the same time of many other serious cases, the complete examination of this patient was not made until after he had been in the hospital for two or three days, and then pneumothorax was discovered. There was nothing in the history by which to fix the time of onset. His health had been gradually failing for some little time. His respirations were quite quiet, and no special symptoms developed; his strength gradually failed, and he died of exhaustion.

On the *post-mortem* examination, pneumothorax was found. The only disease in the whole body proved to be a small caseous cavity near the root of the right lung, and it was this which had ruptured, the perforation being still patent.

Among the cases previously recorded of recovery without effusion, Case 2 is a striking instance of this kind.

This patient, a miner, continued at his work until the nature of the case was recognised by his doctor, when he was sent up to town to me, but he never was able to understand why it was thought necessary to invalid him. Ultimately, the man got quite well, and remained well for many years.

Case 5 in the same series is another instance of the same kind, and that patient was still living, some years after.

Pneumothorax in the Apparently Healthy.—In 1884 I brought[1] forward a case of this kind which was interesting, because it recovered completely without effusion; and I referred to 17 other similar cases recorded by other observers. This list was supplemented with 5 other cases by Dr. Whipham in 1886, and with another 5 by Dr. de Haviland Hall in 1887, making up to that time a series of 27 cases; and to these may now be added the other cases recorded here.[2]

My own personal experience consists of 6 cases (Cases 2, 3, 4, 5, 8, 14), a somewhat remarkable experience, considering the comparative rarity of the conditions.

In the series of recorded cases, one cannot fail to be struck by the great preponderance of males, for there are only two females among the number. The other fact of importance is the age. There was 1 case at the age of 50, another in a person a little older, and another at the age of 39. With these exceptions there were none over the age of 30. All occurred about the time of life at which phthisis is most prevalent, and this is of some importance in considering the nature of the cause.

The Cause.—Out of 17 cases the cause is assigned in 11, and attributed in 4 probably to phthisis, in 5 to emphysema, and in 2 to over-exertion.

Over-exertion is often a very far-fetched explanation, for the pneumothorax frequently did not follow the exertion till after the lapse of some hours or days. But few attacks occur at the time, and in them the exertion was not such as could be described as attended by violent respiratory effort, *e.g.*, the patient may have been going up stairs, running, or even walking quietly. I have already shown that unbiassed consideration of the facts must lead to the conclusion that a healthy lung cannot be ruptured by any respiratory effort which can ordinarily be brought to bear upon it. It follows, therefore, in these cases that, though the patients may appear to be healthy, the lungs cannot be sound.

The lesions, to which the rupture has been attributed, are emphysema, pleural adhesions, or phthisis.

I have already shown that there is really little evidence at all to prove that emphysema can lead to pneumothorax, nor is there any more conclusive evidence in favour of pleural adhesions as such; and many of the adhesions are really associated with and due to tubercular affections in the lungs.

These being the facts, we are forced to the conclusion that phthisis must have been the real cause in the great majority, at any rate, of cases in which the patients appeared to be healthy. This conclusion receives support also from our knowledge (i) that tubercular lesions may be very slight and impossible to detect by physical examination; (ii) that they may be found *post-mortem* in

[1] *Clin. Soc. Tr.*, vol. xvii. 59; also vols. xix. and xx.

[2] To the bibliography given in the above volumes of the *Clin. Soc. Tr.* may be added three other references—Förster, *Arch. f. kl. Med.*, vol. v.; Austin Flint, *Clin. Lect.*, i., 1875; Klemperer, *Deut. med. Woch.*, 1893, No. 25. Waller, *Lancet*, Feb. 8, 1890, records a good case.

persons who have died from other causes, and who had been apparently healthy up to the time of the last illness; and (iii) that some of the patients, who have apparently been healthy at the time of the occurrence of the pneumothorax, present subsequently definite physical signs of phthisis, or give good reasons for the belief that they are phthisical by the occurrence of symptoms, such as hæmoptysis.

Case 15.—In this connection may be mentioned, also, a case in which the symptoms were very slight or even misleading. The patient, a girl of 16, was admitted into the hospital because she had been constantly vomiting for a week. Two days later she was seized with pain in the left side, which distressed her so much that she was constantly shrieking from the pain. She was found to have pneumothorax. There was, however, no dyspnœa, as the shrieking, indeed, would imply. She had not been ill, as far as could be ascertained, before the onset of the vomiting, a week before she was admitted. The total duration of her illness was only eight days, when she died.

Early tubercular mischief was then found at both apices, and the perforation was still patent in the left apex.

We must, therefore, conclude that in these cases of pneumothorax in apparently healthy persons the lungs are really not sound, but are the seat of tubercular disease.

In spite of this, the prognosis in this form is better than in the others, excepting, of course, the traumatic; in other words, a considerable number of cases of pneumothorax in apparently healthy persons recover completely. There is nothing in this fact necessarily incompatible with their phthisical origin, for we know that, even where the phthisis is actively progressing, the pneumothorax may recover, and a very striking instance of this has been recorded (Case 1).

Recurrent Pneumothorax.—Where the first attack of pneumothorax has been recovered from, it may be followed, after a certain interval, by another. The interval may be very short. Thus I have described two cases where the second pneumothorax developed while the patient was under treatment for the first.

Usually the attacks are separated by a distinct, it may be a considerable, interval of time.

I have referred to one instance[1] in which the patient came under observation for his third attack of pneumothorax within the space of a few months.

Finney[2] records an instance of two attacks of pneumothorax occurring in the course of six weeks. The physical signs were obvious enough. The symptoms, though sufficient to keep the man from work, were not severe enough to take him to the hospital. Recovery was complete.

The most remarkable case of the kind I know is that recorded by Mr. Gabb,[3] who attended a lady, aged 56, in what was apparently a fourth attack of pneumothorax. The first attack occurred in 1874 and the second in 1879, in both of which she was seen by Dr. Byrom Bramwell; the third in 1882, and the fourth in 1887. In each case the pneumothorax was on the right side, and recovery was complete.

Austin Flint records a similar case. Beyond the interest and rarity of these cases, I do not know that they possess any great clinical importance.

Double Pneumothorax.—Pneumothorax on both sides at the same time is hardly compatible with life. The recorded cases have invariably been the result of injury; yet it is astonishing how long life may be prolonged. In the two cases to which reference has been made already, the patients lived forty-one and twenty-eight hours respectively.

A remarkable case of this kind is recorded by Bilton Pollard.[3] A boy, 2½ years old, fell on to some railings, which pierced both sides of the chest, making wounds of 2½ to 3 inches long on each side. The lungs were retracted, but were forced out of the wounds on each side with expiration.

[1] *Brit. Med. Jour.*, 1888, July 28.
[2] *Dubl. Jour. of Med. Sc.*, 1898.
[3] *Lancet*, Feb. 17, 1894.

The wounds were cleaned and closed. In three days all air was absorbed from the pleura, and recovery was completed without bad symptoms.

It is interesting in this connection to recall the cases of double empyema in which an operation has been performed at the same time, or at very short intervals, on both sides, as many of these cases have been followed by recovery without grave symptoms.

The only instance of double pneumothorax of spontaneous origin which has come under my observation is No. 8. In this case the first attack of pneumothorax was on the left side; it was followed by serous effusion which required paracentesis, and recovery ultimately took place. The second attack occurred on the opposite side six months later, from which also complete recovery occurred. The patient has remained in good health for many years, and his chest appears perfectly normal.

Dr. Otto Lasius[1] describes a case in which a woman, aged 27, in the sixth month of pregnancy, was seized suddenly with extreme dyspnœa. This was found to be the result of pneumothorax of the right side. The next day she was delivered of an infant, which died a few hours later. Three days after this the dyspnœa became worse, and pneumothorax was discovered on the left side. She lived ten days after the first attack. At the autopsy the diagnosis was confirmed. The left pleura was full of air, but on the right side the pneumothorax was loculated, the pleural cavity being divided by old adhesions into two compartments, which communicated by a narrow channel. The pleura on both sides contained fluid as well as air.

Drasche,[2] out of 318 autopsies of pneumothorax, found pneumothorax on both sides in 11. In most instances the second pneumothorax occurred shortly before death, and was not diagnosed. In 3 cases the diagnosis was made during life.

F., 22, with advanced phthisis, developed a partial pneumothorax on the right side, the pleural cavity being largely obliterated by adhesions. Within twenty-four hours of the first, the second developed on the left side, and was complete, and the patient died twelve hours later.

M., 22, with phthisis, developed pneumothorax on the left side on 15th March, and on 18th April on the right side, and died two hours subsequently.

M., 26, with phthisis, on 11th June developed pneumothorax of the left side, of which he partly recovered. On 24th July, while up, he gave a sudden cry and dropped down dead. He was found to have pneumothorax of the right side also.

DIAGNOSIS.—The diagnosis of pneumothorax in ordinary cases is easy enough. There is the urgent dyspnœa to fix attention on the chest, and the characteristic physical signs to establish the nature of the disease. Dyspnœa of such intensity and suffocating character as that of pneumothorax is never met with except in embolism of the pulmonary artery, or in the impaction of a foreign body in the larynx or trachea; and from these conditions the diagnosis is easy, for whatever physical signs there are affect both sides of the chest, while the displacement of organs, and the other characteristic signs of pneumothorax, are absent.

There are two classes of cases which would naturally create difficulty. First, that in which the symptoms are so slight that there is no indication of any serious trouble in the chest; and, secondly, that in which the physical signs are somewhat misleading.

1. *Symptoms slight.*—In the first group of cases, in which dyspnœa is absent, either because it was never a prominent symptom, as in latent pneumothorax, or because the patient came under observation in a later stage when it had passed off, the diagnosis has to be made by the physical signs alone, but these are usually obvious enough.

Where the condition is overlooked, it is usually because it is assumed, as many of the old writers taught, that pneumothorax could not set in without urgent symptoms. Now that the contrary is recognised as a fact, the number of recorded instances of latent pneumothorax is rapidly increasing.

[1] *D. med. Woch.*, 1891, No. 15. [2] *Wien med. Woch.*, 1900, June 30.

2. *Physical signs misleading.*—In the second class of cases the physical signs may be misleading; that is to say, the characteristic physical signs are absent, and those which are present admit of other interpretations.

Thus the displacement of organs may be absent, or, if present, may be due to other causes.

There are, practically, only two conditions under which a pneumothorax can occur without displacement of organs, viz.:

(*a*) That in which the heart and mediastinum are themselves fixed to the chest walls.

(*b*) Where the lungs are adherent, so that the pneumothorax is only a partial one.

These conditions could hardly be distinguished the one from the other. The first condition, in which the lungs themselves are not adherent, but the mediastinum and heart are fixed, is one which I have never seen.

3. *A large cavity in the lung.*—With a partial pneumothorax the diagnosis from a large cavity in the lung might not be easy. I have seen two or three instances in which a large cavity in the lung had been diagnosed and treated as pneumothorax, but never one of the opposite kind, in which a cavity in the lung has been diagnosed and the disease proved to be one of pneumothorax.

The diagnosis really presents theoretical rather than practical difficulties. It is only with chronic cavities of very large size in the lung that confusion could arise, and such very large cavities are, of course, rare. Of the cases in which I have seen the mistake made, in one of them the whole of the lung was excavated, and in another two-thirds or more of it.

Such enormous cavities may, of course, yield succussion and the bell sounds, and the fluid present may even be defined by percussion, and change its level readily with position.

These phenomena were, indeed, all present in the cases I refer to. Metallic tinkling and amphoric breathing may be also present. Percussion will, however, usually determine the diagnosis, for it is but rarely tympanitic, as in pneumothorax, but yields a flat or boxy note. The side, moreover, is usually contracted and not distended, while the organs, instead of being displaced on to the opposite side, are drawn over to the affected side.

In the two cases in which the diagnosis of pneumothorax was made, in one of which I made the *post-mortem* examination myself, I do not think that, with due attention to the points already referred to, the mistake need have been made.

4. *Increased area of stomach resonance.*—There is another set of cases in which the diagnosis may present difficulties, viz., that in which great contraction of the lung or pleura has taken place on the left side, with the result that the diaphragm has been drawn up high into the chest, so that the area of stomach resonance has been greatly increased. Under some circumstances the stomach resonance may reach high up into the axilla, even almost to the top of it, the percussion note being markedly tympanitic; the bell sound may be obtained; and if the stomach also contain fluid, succussion may be elicited. There will, however, be no displacement of organs on to the right side; they will, on the contrary, be drawn over towards the left; the lower part of the chest will also be flattened and its movements absent, while the upper part will probably give evidence, in percussion-dulness, and other physical signs, of some long-standing mischief.

5. *Complementary emphysema*, or hypertrophy of one lung, has also sometimes been confused with pneumothorax.

In such cases the organs are displaced to one side, and there may be general hyper-resonance on the other. Still, very little care is sufficient to prevent mistake, for over the hyper-resonant lung more or less vesicular breathing is heard, and on the opposite side there is evidence of the cause of the displacement of organs, *e.g.*, chronic pleurisy, or of chronic disease or excavation of the lung.

6. *Subcutaneous emphysema.*—Where there is much subcutaneous emphysema over the thoracic walls, as happens sometimes after a fractured rib, the hyper-resonant note obtained on percussion may suggest the presence of pneumothorax.

In fact, pneumothorax is mentioned as a common result of a fractured rib in all the older text-books, yet it is now generally admitted that it is of very rare occurrence. The diagnosis formerly rested, it would seem, less upon facts than upon a theoretical assumption that because the ribs were fractured, the lungs injured, and subcutaneous emphysema produced, it must necessarily follow that air had passed into the pleural cavity. This assumption is wrong. The absence of pneumothorax may be proved during life by there being no displacement of the heart, and established after death by examination of the body.

7. *General emphysema of the lung* is sometimes stated to cause a difficulty in diagnosis, but this need hardly be, for pulmonary emphysema is a bilateral affection, so that there is no displacement of organs; the breath sounds, more or less modified it may be, are audible everywhere; and the percussion note, hyper-resonant but hardly tympanitic, does not extend in emphysema so low down as it does in pneumothorax. This latter point alone is sufficient for diagnosis in most cases. In pneumothorax the tympanitic percussion note extends right down to the costal arch, and sometimes even beyond it; in emphysema it stops considerably short of this. On the left side this, of course, is not so easy to demonstrate because of the resonance of the stomach; but on the right side it is easy enough, for in emphysema the upper border of the liver rarely stands below the level of the seventh rib in the nipple line, *i.e.*, two inches or so above the costal arch.

8. *Skodaic resonance, i.e.*, the hyper-resonance obtained in front or over the upper part of the chest where there is solid lung or effusion behind, may present more real difficulties, and in certain cases of pneumothorax it is difficult, during recovery, to say whether the hyper-resonant area left is due to relaxed lung or to the continued presence of a little air in the pleura. With skodaic resonance, however, though the percussion note may be quite as tympanitic as in pneumothorax, the breath sounds are audible and of a more or less vesicular character, while, if the side contain fluid, succussion is absent and so is the bell sound, and the fluid does not so readily change its position.

Indeed, with ordinary care in physical examination, it is difficult to confuse skodaic resonance with pneumothorax.

9. *Rupture of the diaphragm.*—This is a condition in which very considerable difficulties may present themselves, for in a case of ruptured diaphragm the onset will be sudden, there will be extreme displacement of organs and considerable dyspnœa. The injury which has caused rupture of the diaphragm might quite as easily have caused rupture of the lung, and the only means of diagnosis is careful physical examination, though this is often impossible to make because the patient is so seriously ill.

It would then be found that, though parts of the pleura were tympanitic, others were dull or not tympanitic, and it is this patchy, irregular resonance of the side which would give the diagnosis.

The following is a case of the kind:—

The patient, a man of about 36, had been run over. He was suffering intense dyspnœa and was deeply cyanosed, and had the usual displacement of organs. The greater part of the pleura was hyper-resonant, but in the axilla in one or two places were patches of irregular dulness, which did not correspond with the lines of effusion, nor, indeed, had effusion had time to form unless there had been bleeding into the pleura, of which there was no evidence. Two points were of importance in this case—the flattening of the abdomen and the defective movement of the diaphragm on that side. The patient shortly after died, and on *post-mortem* examination the stomach, parts of the colon and small intestines, as well as the spleen, were found in the left pleural cavity.

Of course, if the diagnosis could be made with sufficient certainty, and the patient's condition was such as to stand operation, it might be right to open the abdomen, replace the organs, and stitch up the diaphragm. The prognosis could not be worse and might be better, for if left alone such cases invariably die.

10. *Subphrenic abscess.*—The last condition that remains for consideration is what is often called Pyo-pneumothorax Subphrenicus. This is a bad term, for it is not pneumothorax at all, but an air-containing, sub-diaphragmatic abscess.

Subphrenic abscess, when not containing air, is more likely to be confounded with a localised empyema than with pneumothorax. The diagnosis between these two conditions has already been considered under Empyema. In this place it will be necessary to consider only that form of subphrenic abscess which also contains air.

The air may be derived either from the stomach and intestines by a communication between the abscess and these organs, or from gangrenous or putrefactive decomposition in the abscess itself without any such communication. It is the latter condition which is most likely to create difficulty, because in the former, where there is a communication with the stomach or intestines, the air or pus, as the case may be, will evacuate itself by these channels, and there will be no great tension in the abscess, and therefore no great displacement of the diaphragm.

Under any circumstances, it would be from partial pneumothorax, and not from general pneumothorax, that the diagnosis would have to be made, and the difficulties would all disappear on careful physical examination. For there would not be the usual displacement of the heart and mediastinum to the opposite side. The displacement, on the contrary, is that of the diaphragm upwards, and the curves follow the same lines exactly as in simple subphrenic abscess, being highest in the mid-axilla or somewhat farther forwards, and falling towards the front and towards the back. The displacement into the thorax is usually greatest on the right side, because on the left there is no solid organ like the liver upon which the abscess may get purchase from below. The diagnosis, however, on the left side may be even more difficult, because a dilated stomach may yield almost the same physical signs.

THE TREATMENT OF PNEUMOTHORAX. — No one can foresee the occurrence of pneumothorax; all that can be done by way of prevention is to caution those whose lungs are diseased against any violent muscular or respiratory effort.

When pneumothorax has occurred, the mischief is achieved.

All that treatment can then do is (1) to relieve the symptoms which have been produced; (2) to prevent as far as possible the complications which may arise; or (3) if they occur, to deal with them as the circumstances require.

The gravity of pneumothorax is not due to the entrance of air into the pleura merely; for if the air be pure, *i.e.*, not infected, it may be completely absorbed and no inflammation of the pleura follow. The gravity depends, in the earlier stage, upon the sudden embarrassment of the respiration and circulation consequent on the collapse of the lungs, and in the later stages upon the inflammatory conditions which follow.

The treatment may be considered in relation to these two stages, the early and the late.

I. In the Early Stage.

The prominent symptoms of the early stage are shock, pain and distress, and dyspnœa.

The shock is often profound. It must be treated on general principles, and for this purpose the rapidly diffusible stimulants are the most useful, *e.g.*, sal volatile, ether, alcohol, and strychnia, given by the mouth, or, if necessary, by subcutaneous injection.

The pain and nervous excitement should be allayed by sedatives, of which opium and morphia are the most trustworthy. These also may be given by the mouth or *sub cutem*, according to the requirements of the case.

The bromides, chloral, cannabis indica, etc., are too slow in their action to be of use where the symptoms are severe.

It is dyspnœa which is the chief symptom, and this is usually extreme.

In the cases in which dyspnœa is absent, slight, or at any rate not extreme, the treatment should be general, and the less active the better.

The patient should be kept quiet in bed, talking should be prevented, and all muscular effort forbidden.

The minor symptoms that present themselves should be relieved by appropriate means: restlessness and excitement by sedatives, such as bromides and chloral; and pain by counter-irritation, local anodynes, or even a few leeches applied to the seat of pain. Great care should be taken to avoid chill. The case should be carefully watched, first, for the signs of congestion of the opposite lung, and, secondly, for the development of fluid on the side affected.

If the dyspnœa be considerable, something must be done, and that without delay.

The dyspnœa, as already explained, is partly mechanical and partly physiological; mechanical, so far as the pneumothorax leads to the complete collapse of the one lung and the partial collapse of the other; physiological, on account of the embarrassment of the circulation and respiration which this sudden collapse of the lungs produces.

The collapse of the lungs, though the result of their own elastic retractility, is rendered more complete by the increased intra-pleural pressure on expiration.

These mechanical factors in the dyspnœa could, in part, at any rate, be removed if an exit for the air were provided from the pleura. If, then, the dyspnœa be urgent, the side should be tapped and the tension thus relieved.

For this purpose an aspirator is not necessary nor desirable, for the expiratory pressure being in excess of that of the atmosphere, the air will readily escape of itself during expiration, while if an aspirator be used in the early stages of pneumothorax, air will be sucked in afresh from the lung through the original perforation, and the hole be thus kept open. This would be a direct disadvantage, for it is good that the lung should remain collapsed for a while so as to give the perforation time to close and become sealed.

There are objections, also, to the use of a simple cannula, for air will then enter on inspiration as well as escape on expiration, and the risk of some infective substance being introduced from outside into the pleura will be increased. To obviate this objection various forms of valvular cannulæ have been proposed, but there is always a difficulty in keeping them clean. The syphon is the safest and best arrangement, *i.e.*, a cannula with a tube attached, the end of which is placed under water. In this way a water valve is formed, which, while permitting the free exit of air, prevents its return.

Paracentesis almost always gives great and immediate relief, though the relief may be unfortunately only of short duration.

If the air re-accumulate and the symptoms return, another paracentesis will be required, and perhaps even a third, fourth, or more.

For such cases as these, where repeated paracentesis is necessary, it has been proposed to insert a cannula, generally a valved cannula, and to leave it in the side. If such a method were employed, it would be best to use the syphon arrangement already recommended.

I think, however, that to leave a cannula in the chest is bad practice, for it is difficult to keep such cannulæ clean, and suppuration almost always occurs in the puncture round the tube if it be left in long; the cannula then becomes loose, and when it is removed the orifice may not close. An external opening may become necessary, but if so, it should be made deliberately after careful consideration, and not permitted to develop accidentally. If repeated paracentesis be necessary, it is best to tap in the usual way, and choose a different place each time. The strictest antiseptic precautions should always be taken, for they are, if possible, even more important in paracentesis for pneumothorax than for pleuritic effusion, inasmuch as the result of purulent infection of the pleura is so much more serious.

In the cases which require repeated paracenteses, free incision of the side may seem indicated; but this would almost certainly be followed by suppuration, and the case be converted into one of pyo-pneumothorax, or rather of empyema.

If all cases of pneumothorax ended, as a matter of course, in purulent effusion, this would make no difference, and free incision at once might be the simplest and best method of treatment. We know, however, that in some cases of pneumothorax no effusion takes place at all, and that in the rest the effusion is often serous. As the ultimate prognosis of pneumothorax depends largely upon the nature of the effusion, it follows that free incision should be avoided if possible. I should, however, not hesitate to recommend incision if I thought it necessary, rather than run the risk of prolonged dyspnœa. Fortunately the cases in which the question will arise are likely to be few.

A good commentary on what has been said is provided by an interesting case recorded by Dr. Crowdy,[1] in which, after repeated paracenteses, a free incision was made; the effusion was serous, and did not become purulent at all. In the course of three months the incision healed, and recovery was complete.

If the dyspnœa increase in spite of repeated paracenteses, it must depend upon other conditions, which are physiological rather than mechanical, viz., upon the congestion of the opposite lung, and the consequent embarrassment of the heart.

To relieve these symptoms, dry cups may be applied to the interscapular spaces and to the whole back, and this often gives marked relief; or the blood may be detained in the extremities, for example, by an elastic band tied round one or both thighs, or by the use of Junod's boot.

[1] *Brit. Med. Jour.*, 1897, p. 367.

In some cases wet cups may be employed instead of dry; but if there is thought to be an indication for bleeding, the desired result will be better obtained by a venesection.

Indeed we have, in certain cases of pneumothorax, the very indication for bleeding, viz., a right ventricle which is becoming paralysed from sudden over-distension. Where the pneumothorax occurs in a person whose previous health has not been impaired to any great extent, there are none of the general contra-indications which in other cases may render it of doubtful expediency. If bleeding be decided on, a large vein should be opened and several ounces of blood rapidly removed. I have no doubt that life might be sometimes saved by timely venesection, and it is certain that bleeding is not as much employed in these urgent cases as it ought to be.

At the same time, whether bleeding be performed or not, the general strength should be maintained in every way, and the heart stimulated by alcohol, strophanthus, citrate of caffein, or even digitalis, while the general excitement and distress should be allayed by the cautious use of opium or morphia.

II. In the Later Stages.

When the urgency of the symptoms has passed off, the treatment must be expectant. What is to be done chiefly depends upon whether effusion follow or not.

If no fluid form, no local treatment will be required; the air will be in time completely absorbed, the lung re-expand, and recovery in all probability take place, unless phthisis or some other grave disease prevent it.

If effusion develop, the treatment to be adopted will depend upon the nature of the fluid, and this must be determined by the needle.

The effusions must be treated on the usual general lines, the only question to be considered being how far these general principles should be modified in the presence of pneumothorax.

1. Where the Effusion is Serous.

Serous effusions in pneumothorax may spontaneously disappear, and they frequently do if only in small amount. In pneumothorax, however, the conditions are not so favourable for absorption as they are with serous pleurisy, for there is no doubt that the lungs themselves play a very important part in the removal of fluid from the pleural cavity, and where they are so completely collapsed they cannot take their usual share in the process.

Where spontaneous recovery occurs, the fluid may be first absorbed and air alone remain, or the air may be absorbed first, and its place be taken by fluid.

Even a serous effusion should not be left too long, and I think, if after two or three weeks the fluid still persists, it would be better drawn off by paracentesis, preferably by syphonage.

On inserting the needle, care must be taken that the mouth of the cannula is below the level of the fluid, otherwise nothing but air will be withdrawn, and the fluid will remain behind. If the mouth of the cannula be below the level of the fluid, expiration is usually quite sufficient to fill the tube, and no suction is necessary.

If, however, the syphon be not filled in this way, a syringe may be used for the purpose, or the tubes may be filled before the needle is inserted.

In most cases this syphon apparatus is all that is wanted, and answers every purpose. The aspirator is a dangerous instrument to use in pneumothorax unless it be provided with a manometer to show the pressure which is being

employed, and this should on no account exceed, if it should even be allowed to reach, as much as 18 to 24 inches of water. Aspirators are rarely provided with such a pressure gauge. If, by using too high pressure, the lung be ruptured afresh, great mischief may be produced; for if, as is probable, the lung has partly re-expanded, it will become collapsed again, and thus progress be delayed; and besides this there is the danger that with the air some infective material may be sucked into the pleura, and the effusion made purulent.

Great delicacy and caution are necessary in operating upon the pleura in a case of pneumothorax. Even in most skilful hands the aspirator may do mischief, and under ordinary circumstances it should not be used. To employ an aspirator with the object of sucking the lung out, and helping it to expand, is vicious in theory and mischievous in practice.

In all cases of tapping the pleura in pneumothorax, whether for the purposes of exploration or paracentesis, especially, of course, in the latter, care should be taken to prevent the air passing along the puncture to the subcutaneous tissue. With this object, as the needle is withdrawn, the finger should be firmly pressed over the puncture, and a piece of sticking plaster, made sticky in the spirit lamp, should be quickly applied immediately over the puncture, and then a pad strapped firmly to the side, so as to exercise pressure over it. Where the patients cough, as they frequently do after puncture of the pleura, air is likely to be driven along the puncture and to reach the subcutaneous tissue, and in this way extensive surgical emphysema may develop. With the air, especially where the effusion is purulent, it is not unlikely that infective substances will be carried with it and excite suppuration in the puncture, and even beneath the skin, and thus in the end lead to an external opening. The risk of this can be greatly diminished by firm pressure, applied after the puncture in the way described.

2. Where the Effusion is Purulent.

The general principles of treatment of pus in the pleura will, of course, apply. The only question is how far they may require to be modified in the presence of pneumothorax.

As in the case of empyema, three courses only are open. It may be left alone, it may be tapped, or it may be incised.

(*a*) *Left alone.*

Although in empyema cases occur in which the pus is spontaneously absorbed, and cure thus effected, I do not know any instance of the kind in pneumothorax. If pyo-pneumothorax be left alone, the pus will ultimately make its way out, either through the lung or externally. If through the lung, spontaneous cure is extremely unlikely, though it may perhaps occur, as in one of the cases which I have reported. If discharge take place externally, the wound will probably have to be extended, and the result will be the same as if an incision had been deliberately made, but with this disadvantage, that a greater length of time will have elapsed, and the lung, therefore, be in a less favourable condition for expansion.

As a rule with pyo-pneumothorax, a condition of well-marked hectic develops, and if nothing be done, the patient dies ere long, exhausted, and that, too, even if the pus make its way out internally or externally.

It follows, therefore, that to leave a case of pyo-pneumothorax alone gives the patient but little chance of recovery.

(*b*) *Paracentesis.*

Temporary relief can, of course, be given by tapping, but I do not know of any case of pyo-pneumothorax in which paracentesis has led to recovery. The operation is certainly not devoid of risk, for the pus is, as a rule, too thick to be removed by syphonage. Suction, therefore, has to be used, and there is considerable risk that in sucking the pus out the lung will be ruptured afresh, and thus much mischief be done. Besides this, the coughing which follows the operation often forces air, and with it some pus, along the track of the needle, which suppurates. Sometimes a considerable subcutaneous abscess forms, which has to be freely incised, and thus a communication with the chest is made; or, the suppuration may take place beneath the periosteum, and in this way a considerable portion of one or two ribs necrose.

In one case of this kind nearly 6 inches of the sixth rib necrosed, and had to be removed in two successive operations. Although there was not at first a direct communication with the pleura, the abscess ultimately burst in both directions, so that when the rib was removed a very large opening into the chest was left.

If paracentesis be performed, it must be remembered that the object is to remove the pus and not the air, and therefore the mouth of the cannula must be directed to the lower part of the chest, where the pus lies; otherwise air only will escape, and no pus be obtained. Also, care must be taken after paracentesis that pads are applied and firm pressure made over the puncture, to obviate, as far as possible, the objections mentioned; and in order to keep the puncture as small as possible, only a small or medium-sized cannula should be selected.

(*c*) *Incision.*

From what has preceded, it would seem that the only thing to be done for pyo-pneumothrax is to treat it like an empyema, and incise early, and that is the conclusion which I think ought to be drawn. At the same time, most authors express an opinion adverse to an incision in pyo-pneumothorax.

The published statistics of pyo-pneumothorax are very much more unfavourable than those of empyema. For this there must be some good reason. It may be that (1) the disease is more serious in itself, (2) that the lung is more likely to be gravely involved, or (3) lastly, that the methods of treatment differ.

As regards the gravity of the affection itself, one cannot compare all cases of pyo-pneumothorax with all cases of empyema, for the great majority of cases of pneumothorax have their origin in phthisis, while the majority of the cases of empyema do not; and we know that empyema occurring in the course of phthisis, and due to it, is of very much graver prognosis than when it is due to other causes.

That the presence of air in the pleura has really nothing to do with the results is shown by the fact that in tapping empyemata air may escape into the pleura, or even the lung itself be ruptured, yet this air is, in the majority of cases, soon absorbed, and the case runs its ordinary course.

It may be thought that the perforation in the lung, being, as a rule, from a tubercular cavity, will not itself heal, and thus the conditions will be much more unfavourable than in empyema: yet this is not altogether correct, for in many cases the perforation does heal, even in a tubercular cavity, while in some of the cases of pyo-pneumothorax, in which *post-mortem* the hole is found patent, it may be not the original perforation, but one which the pus has subsequently made for itself in finding its way out through the lung.

Putting aside, then, the fact that the lung is more likely to be diseased in pyo-pneumothorax than it is in empyema, there seems no other reason why the air and pus in the pleura should not be treated in the same way in the two cases, so that the more unfavourable results in pyo-pneumothorax must be in part at any rate attributed to the difference in treatment of the two affections. The difference in the statistics of pyo-pneumothorax and of empyema would not be so great if we compared pyo-pneumothorax, not with empyema operated on early, but with those cases which were neglected, *i.e.*, not operated on at all, or only after long delay. The statistics of pneumothorax are drawn from old sources, and there are no recent statistics of pneumothorax treated by early incision. I venture to think that if there were, the results shown would be very different.

The reasons given for the postponement of incision, or even of paracentesis, by older writers are chiefly two: first, that by the compression which the pneumothorax exercises upon the lung of the affected side, the progress of tubercle in it is checked; and secondly, that the tubercular mischief often makes rapid progress when the lung re-expands.

These *a priori* objections are not absolutely correct, and express at the most only a part of the truth; and against these must be set the facts, 1, that tubercle may certainly progress in a collapsed lung, for recent tubercles may be found in lungs which have been long collapsed; 2, that tuberculosis does not by any means always rapidly progress in an expanded lung after its having been collapsed, as is shown in the case of serous pleurisies and in empyemata; 3, that, on the other hand, tuberculosis may develop and progress rapidly in the opposite lung after, and apparently in consequence of, the collapse of the lung on the affected side.

The same arguments were once urged against the tapping of serous effusions which were thought to be tubercular, as well as against incision in empyema, and with as little reason. They were over-ruled by experience. It follows, therefore, that the *a priori* objections should not be allowed to weigh much against the treatment of pyo-pneumothorax by incision.

At the present time pneumothorax is very rarely deliberately incised. If an incision is made, it is generally either because the pus has spontaneously discharged itself externally and the wound requires extension, or because, after paracentesis, the puncture has suppurated.

My own feeling is strongly in favour of the treatment of pyo-pneumothorax by incision. We have no statistics as yet which can guide us in the matter, and we must make them for ourselves; but I believe it is quite justifiable to make a forward step in this direction.

In a case of pneumothorax, as soon as the effusion which has formed is known to be purulent, the question of its removal should be considered. As long as it is merely sero-purulent, and especially if it will flow through a syphon tube, the effect of paracentesis may be tried. If, however, the pus be thick and viscid, and require an aspirator, with more or less suction, the question of incision should be raised. There need be no great hurry in deciding, for it is well to give the perforation in the lung time to close and heal firmly. At the same time it would be wrong to postpone incision too long. There will be room, no doubt, for considerable judgment to be exercised in the choice of the exact time for operation, which would have to be determined partly by the length of time which had elapsed since the pneumothorax, as well as by the general condition of the patient, and the state of the lungs.

The operation should be conducted in the usual way, and there is but one question which remains for consideration, viz., the propriety of washing the side out. In many of these cases which are tubercular, the pus is not only thick, but seems to contain a large amount of mucus, and is very viscid. I think it well to wash out the pleura freely at the time of operation, and to repeat it from time to time if it seem necessary. I have done this in several cases; the result has always been satisfactory, and I have never seen any harm come from it.

In many cases pyo-pneumothorax has come under my treatment only after some time had elapsed from the commencement, and I therefore had but little opportunity of trying what I am here advocating, viz., early incision; but I believe that not only is it the right line of practice, but that it will soon come to be recognised as such, and become the rule. If a pneumothorax be left for months, there is the same prospect, neither more nor less, of curing it by incision, as in a case of empyema left untreated for as long a time, and, therefore, if incision is to be made in a case of pyo-pneumothorax, it should not be postponed too long.

If pneumothorax may be treated by incision when necessary, it next remains to consider when it becomes necessary.

1. *During the early stage.*—It is in the early stages of pneumothorax that the risk to life is greatest, and it becomes less and less as time goes on. This is due to the sudden embarrassment of the respiration and circulation, caused by the sudden collapse of one lung and the congestion of the other. If life be prolonged and time be given, the lungs and heart adjust themselves to the altered conditions, and the urgent symptoms pass off.

To relieve these urgent symptoms in the early stage paracentesis is often necessary, and may have to be repeated. If, after paracentesis, the conditions again became urgent in a very short space of time, so that paracentesis has to be repeated at short intervals, it may be wise to consider well whether the side should not be incised. This would have the effect of giving a permanent vent to the air and relieving all pressure symptoms; but it would have two obvious disadvantages; first, the lung, being partly expanded, would be in a less satisfactory condition for the perforation to heal soundly; and, secondly, an incision would almost certainly end in this case, as in others, with the production of an empyema. It is wise for these reasons to entirely avoid, if possible, a free incision in the early stage of pneumothorax, or, at any rate, postpone it as long as possible. All the relief that is requisite can usually be given by means of repeated paracenteses. However, if paracentesis should fail, I should not myself hesitate, even in the early stage, to lay the side open; and I should be inclined to do this in preference to using any form of valve tube. The only tube of this kind which I should be inclined, under any circumstances, to employ would be a fine trocar connected with a small indiarubber tube, extending, say, to the floor from the bed, and opening under water; in this way a fluid valve would be formed which would permit the escape of the air from the pleura, but not allow any air to pass back. The objection to any of these tubes is that, do what we may, in most cases the puncture suppurates, and sometimes leads to an opening into the pleura which might, perhaps, otherwise have been avoided. I should prefer in these cases repeated paracentesis, and if that failed, I should be inclined to proceed to incision.

2. *During the later stage.*—If the early hours and days of pneumothorax pass over without symptoms of such urgency as to call for incision, the question of incision becomes one which may be considered at leisure, without hurry, and deliberately decided upon.

(*a*) If no fluid form, and the case remain one of simple pneumothorax, the air in time will be absorbed and recovery be complete. This may take some days, or even two or three weeks, but in the end the air will all be absorbed. I have never seen a case of simple pneumothorax in which the air did not in the end spontaneously disappear, and that, as a rule, in not many days. It is well not to interfere with these cases in any way, even by paracentesis; and if, with the view of accelerating recovery, paracentesis be performed, the aspirator should not be used, but the air removed without any suction. If suction be employed, it should be limited to a few inches of water, say ten or twelve at the most. These cases are, however, best left without interference.

(*b*) Where serous effusion develops, so that the case is one of hydro-pneumothorax, it should be treated on the general lines of serous effusion; paracentesis may be performed, but without suction, care being taken to remove the fluid rather than the air. Cases may be cured in this way by paracentesis, but sometimes they cure spontaneously, the air being absorbed and fluid taking its place. Under these circumstances the subsequent treatment becomes that of serous effusion, and it may be that where repeated paracentesis has failed to cause the disappearance of the fluid, incision might become necessary, just as it does sometimes in cases of simple serous pleurisy.

(*c*) Where the effusion is purulent, that is to say, where we have a case of pyo-pneumothorax to deal with, and careful paracentesis fails to give relief, incision will become necessary. The chance of pus being spontaneously absorbed in pyo-pneumothorax is very much less than it is in empyema, and there it is, as a rule, small. Sooner or later incision will become necessary, and there should not be too long delay in deciding upon this operation. The indications for operation in pyo-pneumothorax are, I think, simply the same as those for operation in empyema, and the presence of air in these cases may, I think, be disregarded. What makes the prognosis in these cases less favourable than in ordinary cases of empyema is, that in so many cases of pneumothorax tuberculous lesions in the lung are present, while they may be absent in empyema.

I do not doubt that if in the future cases of pneumothorax are treated on the same general principles as govern our practice in empyema, the results will be much more favourable than they have been hitherto.

Appendix.

I append a case in which I put into practice the principles I am advocating, and with great success, although under many disadvantages, for the pyo-pneumothorax had been left untreated for four months, and the patient was reduced to a condition of serious illness. I add to the account of the case the comments I made on it at the time, as they sum up and bring to a focus the points I have laid stress on.

W. T., aged 34, a blacksmith, was admitted to the hospital under my care on 8th March, 1897, complaining of cough and difficulty of breathing. He proved to have a pyo-pneumothorax of the left side.

His previous history was this: on 2nd November 1896 he was taken suddenly ill while at work, with such sharp pain in his left side that it was unbearable on coughing. He went home to bed, and stayed there for eleven weeks. He was told that he had pneumonia and pleurisy. Shortly after Christmas time, soon after getting up, he had an attack of violent dyspnœa, during which he became unconscious for a short time. He had had two slighter attacks since, and had been very short of breath on any exertion. For the last six weeks the patient had noticed a peculiar splashing sound in his chest as he walked, and he had shooting pains in different parts of the left side of the chest. His cough during this time had been frequent and paroxysmal. There had been much dark, thick expectoration, but no hæmoptysis.

On 4th January the patient went to the Convalescent Home at Sandgate, where he remained five weeks, and while he was there the doctor drew off a small quantity of clear fluid from his chest.

Returning from Sandgate, the patient went to St. Bartholomew's Hospital, where he was shortly after seen by me and recommended for admission. During his illness he had lost two stone in weight, but he lost three during the first part of his illness, and regained one subsequently at Sandgate. There was nothing in the patient's previous history or in the family history which had any bearing upon the case. The patient looked pale and ill, and had evidently lost a good deal of flesh. He was disturbed frequently by paroxysms of coughing, which were occasionally very severe. He presented the signs of left-sided pneumothorax, the cardiac dulness reaching as far as two inches to the left of the sternum.

The diagram shows the limit of hyper-resonance as the patient lay upon the back; in front the voice -and breath-sounds were amphoric in character, and the bell sound audible over the whole hyper-resonant area; behind, the breath sounds were absent at the base and altered in character at the apex, and the bell sound stopped at the limits shown upon the diagram; above and below between the two lines the bell sound and amphoric breathing and voice sounds were present, as in front. Succussion was easily elicited. The lung, it was thought, was adherent at the apex behind. The hole in the lung, in spite of the amphoric breathing, was evidently closed. As a cyrtometer-tracing showed, the affected side was rather smaller than the opposite one.

16th March.—A needle was inserted into the seventh intercostal space behind in the mid-axillary line, and sero-purulent fluid obtained.

The patient had a pneumothorax with sero-purulent effusion. He was very ill, and quite incapacitated for work. He had been invalided for some months and was getting steadily worse.

The question was: How was he to be treated? The fluid might be removed by paracentesis, but experience of similar cases was not encouraging. He could be left alone, but it was pretty evident what this would end in, and that to do nothing was tantamount to permitting the man slowly to die. Free incision seemed a better alternative than this, for it would give the patient, at the worst, a chance, even though so much time had been already lost. Finally, if the side were to be opened at all, the sooner it was opened the better.

Accordingly, after giving the patient the advantage of a few days' rest and feeding in the hospital, I decided to have the side freely opened.

On 30th March a simple incision was made into the side in the sixth space in the posterior axillary line; a large amount of sero-purulent fluid escaped, probably about 4 pints. An examination with the probe, immediately after the operation was completed, showed that the lung was not more than an inch from the chest walls everywhere, so that it had expanded at once after the operation. Directly the side was opened the patient began to cough, and coughed violently. This might have been due to the irritation of the tube which was inserted, but was more probably owing to expansion of the lung. The patient took chloroform well, and bore the operation satisfactorily. A few minims of laudanum were given on the tongue to check the cough.

31st March.—The patient had been comfortable after the operation, except that once or twice during the night he seemed a little faint, and required some brandy. The next morning, when the wound was dressed, air was found to enter the whole of the left lung, and friction sounds were audible not far from the seat of incision. The patient complained of a good deal of pain in the left side, which was probably connected with this friction.

The discharge from the side was very free, so that for the first few days the patient had to be dressed twice daily. After this the discharge decreased greatly, and it was sufficient to dress the side once daily.

On April 7th the discharge became very offensive, some small pieces of sloughing tissue were removed from just inside the wound, and it was thought that a spot of bare bone was felt by the probe. The side was washed out with a weak solution of iodine and with boracic acid; the discharge rapidly lost its offensive smell, and the spot, where it was thought the bone was bare, healed over.

From this time the discharge rapidly diminished in quantity, and on 25th April it was about 2 or 3 ounces daily, and it continued to this amount for a good long time. Examination with the probe showed that the lung was in contact with the chest walls all round the incision, but that the track in which the tube lay extended for a distance of about 5 or 6 inches straight into the chest in the direction of the spine.

On 19th May the patient began to complain of a good deal of pain in the chest, sometimes round the wound and sometimes a little deeper, but no cause for it could be ascertained. The sinus had, up to the present time, been regularly washed out with carbolic acid solution. This was now stopped, and the pain was a little relieved ; but as the pain continued, various experiments were tried with the tube, as it was thought to be connected with some irritation produced by it. The tube was shortened, but the pain was not much relieved.

On 16th June, on probing, a tender spot was found far back, and it seemed to me that at this spot the tube pressed on an intercostal nerve, and thus caused the pain. The tube was removed, and a longer one of smaller diameter inserted, and from this time the patient suffered no more pain. On 25th June it was stated that the patient felt very much better ; he had been up some little time, and he managed to go up and down stairs moderately well ; the amount of discharge was still about the same, but not more than could be accounted for by the irritation of the track of the tube. On 8th July he had some pain in the leg, which proved to be a little periostitis. This was rapidly cured with belladonna and glycerine, and gave no further trouble. The discharge was now not more than about an ounce in the twenty-four hours ; the patient was up daily, and gaining strength and weight.

On 12th October the patient's weight was 10 st. 11 lbs. ; the discharge was very slight, but, as it did not completely cease, the sinus was injected with creasote dissolved in olive oil (25 per cent. solution). Although this caused a good deal of smarting, it seemed to do a great deal of good. By the end of the month the tube had been considerably shortened, and by 1st November it was out, and the wound completely healed. On 2nd November I made the following note :—"The patient is in excellent health, looks fat and well, and says he never felt better in his life, and feels quite able to go back to his work, that of a blacksmith." The side was a good deal contracted, but no very large amount of deformity had been produced. The vocal vibrations and vocal resonance were felt right down to the base behind. The percussion, at the lower parts especially, was still somewhat impaired ; the breathing was audible over the whole side, though, of course, not as loud as on the opposite. The temperature was normal. The patient had gained 2½ lbs. during the last week.

As regards the temperature, all that need be said is this : that at the time of admission and up to the date of operation, the temperature was normal, or rather somewhat subnormal, and remained subnormal for the first few days after the operation ; it then began slowly to rise, and remained moderately hectic, varying daily from about 102° to 99·5° for about a week or ten days, after which it became subnormal again and continued so, with few occasional rises for a day or two, throughout.

The subsequent history of the case is as follows :—

The patient was sent into the country, and on his return, in the beginning of 1898, looked strong and well. He obtained a good berth as caretaker in an office, and remained for some weeks quite well. Then a little discharge made its way out in the seat of the old incision, which was opened again and a small tube inserted. The track was examined and found to be a narrow sinus, not more than a quarter of an inch wide, leading 3 to 4 inches into the chest, in the mid-axillary direction. It discharged a drachm or two only of watery sero-pus in the twenty-four hours.

The pus was subsequently examined, and found to contain tubercle bacilli, so that it was evident that the sinus either communicated with a tubercular cavity in the lung or had itself become tubercular. Injections of creasote in sterilised oil were tried, but without effect, and the sinus continued to discharge. Except for this fistula the chest had completely recovered, and good breathing was audible over the whole side.

The patient came twice a week to the hospital to be dressed and seen. He continued in excellent health and general condition, was fairly active, and did his work to the full satisfaction of his employers, though his breath was short on exertion.

In the summer of 1899 he began to lose flesh and strength, but no definite physical signs of phthisis were found until the late autumn. Then the right apex showed slight evidence of tubercular mischief, but the constitutional disturbance was out of proportion to the physical signs. He rapidly lost flesh and strength, and at the end of the year died of phthisis, the fistula and the discharge from it continuing unaltered to the end.

The pneumothorax had been completely cured, but the tubercular disease which had caused it continued, and though it remained in abeyance for more than a year, ultimately recrudesced and killed him.

Remarks.—Here is a case of pneumothorax of several months' duration with a purulent effusion. The patient had been under the care, shortly before I saw him, of an eminent authority, who had dismissed him as incurable ; yet, as the result has shown, the case was not incurable when treated on general principles.

We may ask why are such cases as these regarded as incurable? Why are they left alone, that is to say, left to die, without the chance of recovery being given them which surgical interference might provide? The answer is—because of certain theories which are commonly accepted and frequently acted on, and which I believe to be erroneous. These are—

1. That where the lung has been collapsed and compressed for some time, whether by air or fluid, it will form adhesions, and be bound down and be thus incapable of expanding again, so that if the side be opened, a large cavity will be left, which cannot contract, and can only heal by the falling in of the ribs on one side, and by granulating up on the other. If this theory were true, of course operation would be undesirable, for there would be a chronic cavity left which could not close, and the patient would suffer from the results of prolonged suppuration, and die probably of exhaustion or of amyloid disease; this is what is stated commonly to occur.

Now, is it correct to assume that when the lung has been compressed in the way I have described, it must necessarily be bound down and be incapable of expansion? Can we make this a general statement or general rule of practice and act upon it? Certainly not. Of course it may be admitted at once that in many cases where patients have died after long-standing empyemata, or pyo-pneumothorax, or sometimes even with serous effusion, the lung has been found tightly bound down by dense adhesions, and therefore has been incapable of expanding and healing; but it must be remembered that these cases have been left alone without operation, and it does not follow that if some operation had been performed in early times the lung would have been bound down in that way.

We must also admit that there are cases in which, very shortly after an inflammation of the pleura has taken place, as, for instance, with a common serous pleurisy, the lung has contracted adhesions enough to considerably hamper its expansion. These cases, however, again are quite exceptional, and we have daily evidence of the fact that the lung may be collapsed for a considerable period of time by serous effusion, and yet not contract these adhesions. Though the plea that these adhesions were likely to form was used as an argument some years ago in favour of the early performance of paracentesis in serous effusion, experience has shown that this fear is not well grounded.

That the lung is not bound down, even after a very considerable lapse of time, both in serous and purulent effusions, I will quote three or four cases to prove.

A young man, aged about 24, had been discharged from the army in July because he had a discharging empyema, which had made its exit through the side and also through the lung, so that he was expectorating about a pint of pus daily, and losing about a similar amount through the opening in the side. He came under my observation six months later, and as he was in good general health, I decided to perform an operation upon him before his health began to fail. Accordingly a free counter-opening was made low down in the posterior part of the axilla, so as to provide free drainage.

Within about a week from this time all expectoration through the lung had ceased, and within five weeks from the time of operation the wounds were closed and healed. The lung had expanded well, and breathing was audible over the whole side. The patient came to see me for some time after that, and I know that he remained in excellent health for some months as long as he was under my observation, and I have no reason to believe that his health suffered afterwards.

Some years ago I read to the Medical Society of London an account of the case of a lady who was suffering from a large right-sided serous effusion which had been deliberately, and after consultation, left alone for a period of eighteen months without even once being tapped. I tried paracentesis several times, and when this failed, I ultimately decided to have the side laid freely open, and the case treated as if it had been one of empyema. Paracentesis had shown me that the lung was not bound down, as I had at first had reason to believe, for after each paracentesis it came out into contact with the ribs. I had hoped, however, after the repeated paracenteses, that the lung would have formed adhesions with the costal pleura, but, to my surprise, when the side was opened no adhesions were present, and the lung collapsed completely. However, this made no difference in the ultimate result, for the lung rapidly re-expanded and came into contact with the chest walls, and formed adhesions there. Soon nothing was left but the track in which the tube lay, and ultimately this also healed.

This case shows, first, that the lung in a case of serous effusion may be collapsed for at least eighteen months, and still be capable of ready re-expansion; and, secondly, that even after repeated paracenteses no adhesion need necessarily take place between the two layers of the pleura.

I have recently had another case of the same kind in a man who had been allowed to have a serous effusion, not operated upon, for about twelve months before he came under my observation. With him I tried also the effect of repeated paracenteses, but, encouraged by the experience of the previous case, after a few paracenteses I decided to have the side opened. Accordingly the side was opened, the fluid let out, and a drainage tube inserted. Within a week from the time of operation the lung was in contact with the chest walls over the whole side, and nothing was left except the long sinus in which the tube lay.

In the case which is the subject of this appendix, although the lung had been compressed for some months, within a week after the incision had been made in the side, the lung was in contact with the chest wall over the whole side, and the pleural cavity closed except for the sinus in which the tube lay.

We have, therefore, a series of cases—one of empyema, two of serous effusion, and one of pneumothorax—in which, in spite of the lung having been compressed by effusion for a considerable period of time, it was not bound down, but was capable of ready re-expansion when placed in a position to expand.

These are extreme cases; and although, on the other hand, it must be admitted that the lung may form adhesions which may bind it down quite early in a case of pleurisy, still there is no evidence that this always occurs, or even that it is the general rule; on the contrary, the evidence all points the other way. If this theory be unsound, the practice based upon it will be wrong.

2. A second theory, which is commonly stated in text-books and is very widely accepted, and which has been often used as an argument against surgical interference, is that the compression of the lung by effusion or air, as the case may be, checks the progress of tubercle in it; so that though the presence of effusion or air in the pleura is an evil, it is outweighed by the advantage of checking the development of tubercle in the lung, and, therefore, no operation should be performed.

This again is, I believe, a completely erroneous theory, though the statement is commonly made and has many distinguished names to support it; still I do not know that it rests upon any conclusive evidence, but remains a matter of opinion only; indeed, what evidence there is leads to the opposite conclusion. It is not the case that the compression and collapse of the lung do necessarily check the development of tubercle in that lung, for tubercles of recent formation

may be found in the lung which is collapsed, of a date, as far as can be judged, subsequent to the time when the lung became collapsed. Certainly the collapse of one lung is not infrequently followed by the rapid development of tubercle in the other.

The same theory was used at one time as an argument against the performance of paracentesis for serous effusion, and no doubt it was under the influence of this theory that the two cases I have recorded were deliberately not interfered with; but, as a matter of fact, at the present day this theory does not really affect our practice at all in respect of pleural effusions. They are tapped, and, I believe, rightly tapped, without any consideration as to the effect the removal of the fluid may have upon tubercle in either the one lung or the other.

In the case of empyema, again, we have almost forgotten that such a theory ever had any influence at all. No one nowadays thinks of leaving an empyema alone because of some possible effect it might have upon tubercle in the lung.

I maintain that these theories should have no more weight in relation to pneumothorax and its treatment than they have in relation to the treatment of empyema or of serous effusion.

3. Another argument that is used against the treatment of pneumothorax by free incision is that experience shows that the results are unsatisfactory. "Leave pyo-pneumothorax alone, because the patient will die any way, and operation will only accelerate that result," used to be the teaching of not many years ago; and, indeed, on this point Fagge, who can hardly even yet be regarded as antiquated, was very precise.

"Operate only when driven to it" was another form the advice sometimes took. What would be the result upon our statistics if the same were applied to empyema, and if we never opened a side until the empyema was pointing, and never touched it even then, if the empyema by good luck would discharge through the lungs?

A question of this kind must be settled by experience, and we cannot take the results of some years ago as a guide to practice at the present day in pneumothorax any more than in empyema.

In this respect, namely, the operative treatment of pneumothorax, we have to make our own experience over again; we cannot be guided by the experience of many years ago, especially when we remember that the statistics are based upon the cases of pyo-pneumothorax which were never operated upon unless operation became absolutely imperative.

I think I have shown, therefore, that there is no valid theoretical reasons why sero-purulent and purulent effusions in pneumothorax should be treated in any different way from sero-purulent or purulent pleurisies, and that the presence of air should not make any material difference.

Of course it is quite true that in the majority of cases of spontaneous pneumothorax the cause is a tuberculous cavity near the surface of the lung; but these ruptures frequently heal, and there is no reason why, when the rupture has firmly healed, the lung should not come into contact with the chest again, and the condition of the patient return to that which existed before the pneumothorax occurred.

Physicians have been in the past, it is true, too much inclined to leave things alone, though I do not think we can say the same of the present day, and it will be something in pneumothorax if we can brush aside the erroneous theories which have hitherto given an excuse for avoiding surgical interference.

73. AFFECTIONS OF THE DIAPHRAGM.

Affections of the diaphragm vary in gravity in proportion to the degree with which they interfere with its action in respiration. In lesser degrees some shortness of breath only is the result; while in extreme cases, such as complete paralysis, the dyspnœa is severe.

The affections of the diaphragm may be roughly placed in three groups, according as the primary cause is mechanical, muscular, or nervous in nature, but, of course, in many the cause is mixed.

I. Mechanical.—In the mechanical group the muscle is prevented from acting as it ought by being thrust into the thorax or into the abdomen respectively; downwards by air or fluid in the pleura or by inter-thoracic new-growths; upwards by peritoneal effusion, abdominal tumours, tympanites, subphrenic abscess, tumour abscess or hydatid of the liver, etc.

When the cause is of short duration and removable, as with fluid in the pleura or peritoneum, the diaphragm quickly resumes its action. Where the displacement is considerable and of long standing, nutritive changes occur in the diaphragm of the same kind as are met with under similar conditions in the abdominal muscles.

The muscular tissue then degenerates and atrophies, so that the diaphragm may become thin and almost transparent. In such cases the muscle would be long before it recovered its nutrition and regained its normal action.

Fig. 164.

Section of diaphragm to show the wide lymphatic spaces. Above is the edge of a sarcomatous growth, which is seen extending along some of these spaces into the diaphragm. (From a preparation in the collection of Dr F. W. Andrewes.)

II. Muscular.—In the muscular group the muscular defects are almost always secondary, as in the common instances just referred to.

Of primary lesions of the muscles of the diaphragm little is known.

Wherever the serous membranes which clothe the diaphragm on either side are the seat of disease, the lesion is almost certain to involve the diaphragm beneath, for structurally the connective tissues of the diaphragm and the lymphatics it contains are directly continuous with those of the serous membranes on either side of it. So close is the connection that infective processes, whether of the nature of inflammation or new-growth, pass readily through the diaphragm from the one

side to the other, in this way not unfrequently involving the other serous membrane without the diaphragm itself presenting any obvious lesion.

In acute inflammation the lymphatic spaces may be found infiltrated with small cells almost to the extent of interstitial suppuration, while the muscle cells are granular and degenerating.

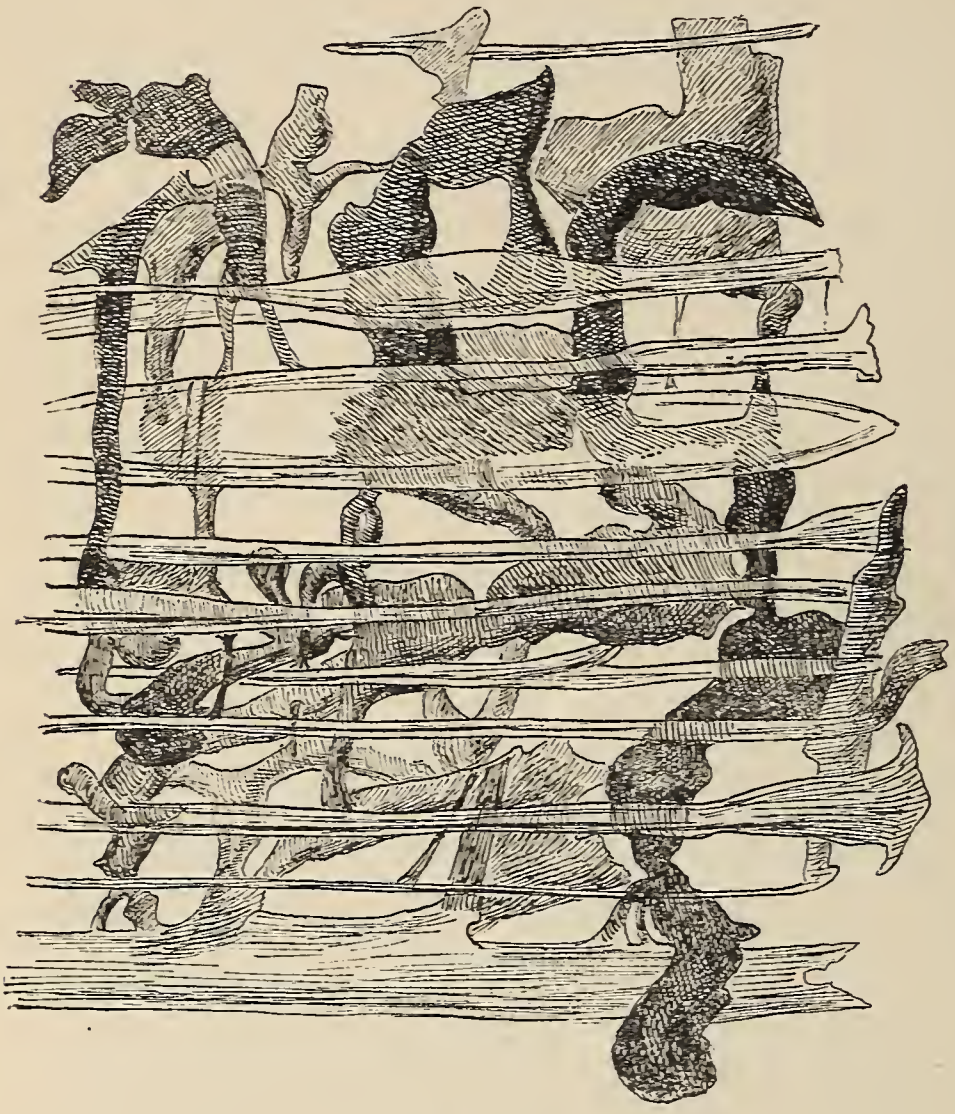


Fig. 165.

Lymphatics of the diaphragm infected, showing the rich network between the muscular bundles. (*Klein.*)

In the case of chronic inflammation of either serous membrane covering it, the diaphragm itself becomes involved, and a sort of cirrhosis is produced, which compresses the delicate muscle-cells within its meshes and causes their atrophy. In extreme cases the muscular tissue may almost completely disappear and a mass of fibrous tissue take its place, extending from the one surface to the other.

III. Nervous. — The most important and interesting group is that in which the cause lies in some interference with the nervous mechanism of the diaphragm.

This may be effected in three ways: first, by inhibition; secondly, by irritation, so that spasm is produced; and, lastly, by paralysis.

1. *Inhibition.*—Where, as a consequence of inflammation of the pleural or peritoneal surface, respiration causes pain, the action of the diaphragm may be inhibited. If the inflammation be unilateral, the movements on the affected side may be abolished while they continue on the opposite side. This is, no doubt, protective, and similar to the inhibition of the peristaltic movements in the intestines observed in peritonitis.

What is called *hysterical paralysis* of the diaphragm might better be described as cerebral or hysterical inhibition, for the diaphragm is, in these cases, hardly ever paralysed; but though immovable at times, especially when the patient is under observation, it moves at other times—for instance, during sleep.

2. *Irritation or Spasm.*—Spasm of the diaphragm is rarely met with as an isolated phenomenon, except in some rare cases of hysteria. Usually it is a part of a general inspiratory spasm, and, as such, occurs to an intense degree in strychnine poisoning, hydrophobia, and tetanus, in which diseases it plays so serious a rôle.

The two commoner and comparatively unimportant instances of spasm of the diaphragm are Paroxysmal Sneezing and Hiccough.

Paroxysmal sneezing stands in close relation with asthma and hay fever, in which connection it has already been dealt with. Hiccough requires further consideration.

3. *Paralysis.*—This is a subject of great importance, and must be dealt with specially.

HICCOUGH.

Hiccough is due to the sudden momentary contraction of the diaphragm, for which the glottis and vocal chords are unprepared, so that the air suddenly drawn into the larynx catches the parts only partially dilated, and thus causes the short noisy croak which is so familiar.

Hiccough is probably due to direct irritation of the terminations of the phrenic nerve, and is frequently produced by the swallowing of irritating food, *e.g.*, very hot or peppery soup.

It is a common symptom of flatulent distension of the stomach. In most cases it is the result of dyspepsia. The attack is then usually only of short duration, lasting a few minutes it may be, and only rarely more than an hour.

Hiccough arising in the course of any acute abdominal affection is a sign of the gravest significance. Thus with acute peritonitis, acute tympanites, or obstruction of the bowels, the appearance of hiccough indicates almost invariably a fatal result.

Strange to say, with the ordinary inflammation of either the peritoneal or pleural surfaces of the diaphragm, hiccough is far more often absent than present, inhibition being produced rather than spasm. Indeed, it always seems to me far more difficult to understand why it is so often absent, than to explain its occurrence.

With pleurisy it is still rarer than with peritonitis, and of far less serious significance; yet it may be very distressing, and by causing pain and preventing sleep, increase the gravity of the case.

It may arise in the course of acute pneumonia, and is then a most serious complication, for it may be very persistent, and obstinately resist treatment.

In a case of well-marked, but not extensive or otherwise severe, pneumonia, hiccough set in about the third day, and continued till the crisis on the sixth, occurring every few minutes night and day, entirely robbing the patient of sleep, and causing great exhaustion. Morphia gave some relief, but the other remedies tried proved useless. With the crisis the hiccough terminated; but when convalescence seemed established, on the second day from the crisis, the patient was suddenly seized with severe dyspnœa, probably from the detachment of a cardiac clot, and died rapidly.

Hiccough is sometimes referred to central irritation, and is thus described in the course of hydrocephalus, meningitis, epilepsy, and hysteria. It is, however, rare in such affections, and is, I believe, even then due rather to peripheral than to central irritation, and to be attributed, as in other cases, to the flatulent distension of the stomach and colon

I do not know of any instance in which hiccough has been produced by pressure or irritation of the trunk of the phrenic nerve in its passage through the mediastinum.

Hiccough, of course, interrupts regular respiration and articulation, and, when severe, may cause great pain along the tips of the ribs, at the points of insertion of the diaphragm. The slight forms last but a few minutes; the more severe may last for days, or even weeks, and the attacks are liable to recur.

Treatment.—For ordinary cases the common popular remedies are to hold the breath for a time, to sneeze, sip water and swallow it slowly, to divert the attention, or by a sudden noise or fright to make the patient start.

When flatulence is the cause, the treatment of that condition will remove the symptom, and with the cure of the dyspepsia the tendency to hiccough will vanish.

In the more severe cases, hot applications and counter-irritants may be applied to the pit of the stomach; faradization or galvanism may be employed to the epigastrium, or along the course of the phrenic nerve; the various sedatives, especially the bromides, may be given, chloroform or ether inhaled, and opium administered by the mouth, or morphia *sub cutem*.

PARALYSIS.

Paralysis of the diaphragm may be *central* or *peripheral*; *central* in the medulla or nuclei of the phrenic nerve, as in bulbar paralysis or broken neck; *peripheral* in the trunk of the phrenic nerve or in the nerve endings, of which diphtheritic palsy is the best example.

It is not necessary to discuss all the modes in which nervous affections cause paralysis of the diaphragm, for this would be more appropriate to a treatise on diseases of the nervous rather than of the respiratory system.

As regards the severity of the symptoms produced, much depends upon whether one side of the diaphragm be affected or both.

With unilateral paralysis, the symptoms are comparatively trifling; but with bilateral, *i.e.*, complete, paralysis, they are severe, and may lead rapidly to a fatal result.

The condition is easily recognised by the peculiar alteration in the respiratory movements. The respirations are accelerated, shallow, and entirely costal; the abdominal movements are inverted, *i.e.*, the epigastrium and abdomen generally are retracted on inspiration and protruded on expiration; the costal arch is widened; and on inspiration the lower ribs move unduly freely. In extreme cases, *i.e.*, where there is much dyspnœa, the contrast on inspiration between the undue expansion of the lower part of the thorax and the recession of the epigastrium is very striking.

The diaphragm stands very high, and, *post-mortem*, may be found to reach the third intercostal space.

Though the chief difficulty is with inspiration, expiration is also impaired, partly because the chest is so incompletely filled with air, and partly because in all acts of forced expiration the contraction of the diaphragm itself is necessary to give direction to the straining.

For similar reasons the voice and cough are weak, and speaking an effort.

Dyspnœa is a prominent symptom in the later stage, but is not due to the paralysis of the diaphragm only, but to the secondary changes which the paralysis leads to in the lung, viz., collapse of the lower parts and congestion of the rest.

To keep the lower parts of the lungs fully expanded is one of the chief uses of the diaphragm. If this fails, especially if the diaphragm be forced upwards into the chest by the unopposed action of the abdominal muscles, the lower parts of the lungs become collapsed and airless, and the rest of the lung congested. The respiratory capacity is thus greatly reduced. In time the lungs may give out, and signs of congestion, *i.e.*, bronchitis, appear. So soon as this occurs, the symptoms become rapidly aggravated, for, owing to the weakness and insufficiency of coughing, the exudation cannot be expelled from the tubes, and soon chokes the patient.

In diphtheritic paralysis it is not, as a rule, the diaphragm alone that is affected; the muscles of the glottis and fauces are more or less paralysed too, so that food and secretions pass into the air-tubes, causing the bronchitis and pneumonia, of which so many patients die. Moreover, the intercostal muscles are often

involved as well, and thus general respiratory paralysis ensues, which must eventually end in suffocation.

The condition of the patient with well-marked diaphragmatic palsy is pitiable in the extreme. The sufferer lies prostrate, feeble, choking with the secretion which the feeble, ineffectual cough does nothing to relieve; the dyspnœa gradually increases, and cyanosis becomes marked; but fortunately the suffering is soon cut short by unconsciousness or by sudden cardiac failure.

Prognosis.—Unilateral paralysis is not of itself of any great importance.

The prognosis of bilateral paralysis depends upon its cause. When the paralysis results from some disease itself incurable, as in spinal and bulbar lesions, recovery is hopeless, though by careful treatment the end may be postponed for a time.

The prognosis in diphtheritic paralysis is grave, not so much because the diaphragm is paralysed, as because the paralysis of the diaphragm is, or soon becomes, associated with other paralyses; if the intercostal muscles be involved, respiratory movements are impossible, and suffocation follows; paralysis of the fauces and glottis permits the passage of food and secretions into the air-tubes, and leads to fatal pneumonia; while in other cases death occurs suddenly from syncope.

Treatment.—The patient should be always fed through a tube, passed well down the œsophagus.

If the fauces are involved, and the pharyngeal muscles paralysed, the secretion, which accumulates in the pharynx and back of the mouth and causes much distress, should be frequently removed with sponges, and belladonna given to check the flow of saliva.

The strength must be maintained, not only by liberal feeding, but by stimulants and strychnia, to which caffein or digitalis may be added, to support the heart. The most valuable combination for these conditions is caffein and strychnia.

The aëration of the blood should be assisted by the administration of oxygen frequently, night and day. Oxygen is the best and most rational remedy for these cases. It should be given as much to prevent, as to relieve, cyanosis, so that its use should not be too long delayed.

Above all things, hope should not be abandoned, for, with dogged persistence, even desperate cases may be saved.

74. DIAPHRAGMATIC HERNIA.

When a hernia develops through the diaphragm, some of the contents of the abdomen pass into the thorax. There is no instance I know recorded in which a hernia of the lung has developed into the abdomen.

The holes in the diaphragm are either traumatic or congenital.

Lacher's[1] statistics show the relative frequency of these two groups to one another to be 4 to 5, for of 267 recorded cases, 117 were congenital and 150 traumatic. This proportion is probably not accurate, for so many of the traumatic cases remain unrecorded.

A third group is sometimes made, and is described as *acquired*. The hernia is then supposed to develop in some of the weak spots in the diaphragm, *e.g.*, at the base of the xiphoid cartilage right and left, at the attachment of the diaphragm, or at one or other of the normal openings through the diaphragm, for the œsophagus, vena cava, or even splanchnic nerve, notably the first.

[1] *Arch. f. klin. Med.*, 1880, xxvii. 268.

This group is of little importance, for the cases are extremely rare, and many of them far from conclusive. It need not be further considered.

Side.—Diaphragmatic hernia of either kind is much more common on the left side, in the proportion of about 5 to 1, owing doubtless to the presence of the liver on the right side, which not only diminishes the chance of rupture of the diaphragm, but when there is a rupture, prevents the abdominal viscera from passing through the rent.

Sex.—The traumatic form is much more frequent in men, 5 to 1, owing to their greater exposure to injury; the congenital form is equally common in either sex.

Organs displaced.—Almost any of the abdominal organs may be found in these herniæ; most frequently the stomach, spleen and colon, but only rarely one organ alone.

According to Lacher, hernia of a single organ does not occur more frequently than 1 in 5 (53 out of 276 cases). In the rest two or three organs are displaced and in all sorts of combinations.

The following figures show the frequency with which the different organs were found:—

Stomach,	in 151 cases.	Duodenum,	in 35 cases.
Colon,	145 ,,	Pancreas,	27 ,,
Small intestine,	83 ,,	Cæcum,	20 ,,
Liver,	45 ,,	Kidneys,	2 ,,

In a few instances (about 1 in 5, Lacher) the hernia is contained within a sac; but this is not important enough to justify the division of these herniæ, as Peacock proposed, into True and False, *i.e.*, with and without a sac.

Congenital.

In the early embryo the pleural and peritoneal cavities are continuous; they become separated subsequently by a thin membrane, the future diaphragm, which grows forward, between the lungs and the Wolffian bodies. In this membrane development may be arrested, with the result that the diaphragm may be deficient in whole or in part.

This condition is usually associated with other congenital defects, such as cleft palate, webbed fingers, etc.

Extreme congenital defects of the diaphragm appear to be incompatible with life, and are, therefore, only met with in still-born children, and belong to teratology rather than to medicine.

Even with less extreme defects the infants rarely live for more than a few hours or days.

When the defect is small, life may be prolonged for some years, and cases are recorded in which the patients have reached middle life.

Thus the well-known case of Astley Cooper's occurred in a man of 49, and another at the same age is recorded by Hale and Goodhart.

Peacock[1] refers to several instances in younger people, at the ages of 17, 22, 24, 28 and 40 years.

A very extraordinary case is recorded by Turner[2] in a youth of 19 years of age, who died of pyæmia, consequent on mastoid disease; the condition was quite unsuspected during life, and discovered only on the autopsy. Nearly the whole of the large and small intestines, together with the spleen, were found in the left side of the chest, the lung being flattened, like a pancake, against the spine. The hole in the diaphragm was 1¼ inches in diameter, and was occupied by duodenum and colon. The abdomen contained an enormously dilated stomach, the pyloric orifice lying in the pelvis behind the bladder.

[1] *Path. Soc. Trans.*, xiv. 149.

[2] *Lancet*, June 14, 1890.

Traumatic.

The traumatic cases fall into two groups, according as they are associated with an external wound or not.

1. In those with an external wound, as, for instance, a stab or laceration, there are often severe injuries to the blood vessels or other viscera as well, which are of more importance than the mere rupture to the diaphragm, and mask it, so that it is often undiagnosed.

2. In those without any external wound, which are the result, for instance, of a fall or a crush, the rupture of the diaphragm is sometimes the only serious lesion; even then the symptoms are often misleading, so that some other diagnosis, *e.g.*, pneumothorax, is more likely to be made.

Symptoms.—The symptoms are at first those of shock, from which many patients die.

After the shock of the injury is recovered from, the symptoms may be either *thoracic* or *abdominal*.

Thoracic Symptoms.—Of these the chief are dyspnœa and pain.

Pain is rarely severe, and not in any way suggestive. It is often distension or tension over the whole side that is complained of, rather than actual pain in any particular part.

Dyspnœa becomes sooner or later severe, but frequently does not set in as soon or reach as great a degree as would be expected from the conditions which are found *post-mortem*.

The dyspnœa depends, as in pneumothorax, chiefly upon the collapse of the lungs, which, on the affected side, is produced by the hernia, and on the opposite side by the displacement of the mediastinum; but its severity varies with the rapidity with which this collapse has been produced. In many cases, though the rupture takes place at a given time, the abdominal organs do not all enter the thorax suddenly at the time of the rupture, but make their way up gradually.

Another factor in the production of the dyspnœa is the presence of fluid in the pleura; either blood, due to hæmorrhage at the time of the accident, or inflammatory effusions, serous or purulent, developing subsequently.

Many of these points are well illustrated by the following case recorded by Dr. Brinton,[1] which is an excellent example of the traumatic group.

A healthy man, 32 years of age, was walking along the street, when a case of machinery fell on him. He was brought into the hospital with a compound fracture of his tibia. He appeared to have received no other severe injury. He complained only of a little soreness in his chest, but no injury was detected there.

The next day he complained of some shortness of breath, and his respirations were rapid, 44. The heart was found beating on the right side, and as the left was hyper-resonant, pneumothorax of the left side was diagnosed.

The day following, the respirations were 48, and the dyspnœa severe; the abdomen was not distended, but flatter than normal; the bowels had not been open since the injury.

The day following, the patient was very cyanosed; the heart still more displaced, and reached as far as 2 inches to the right of the right nipple line; metallic tinkling was heard at the left apex; and the bell sound in front, between the second and fifth intercostal spaces. A trocar was inserted, but no air escaped.

In the evening the patient died, about seventy-four hours from the time of the accident.

Post-mortem examination.—The diaphragm was found ruptured on both sides; on the right side the rent was small, and was closed by the liver; on the left, the rupture, 8 inches in length, extended from the œsophageal opening nearly to the false ribs. Through this rent various abdominal organs had passed into the thorax, viz., the stomach, the transverse colon, the great omentum, spleen, and several coils of small intestine. Besides these organs, the pleura also contained one pint of blood.

[1] *St. Barthol. Hosp. Rep.*, 1883, p. 285.

It is to be observed that although the rupture of the diaphragm must have occurred, of course, at the time of the accident, there is a special note to the effect that the patient showed signs of no other injury except the compound fracture of the leg. It was not till some hours after admission that the respirations were observed to be hurried; and not until the day following that anything which could be called dyspnœa developed. From this time the dyspnœa rapidly increased, till the patient died of suffocation.

The physical signs were positively misleading, for not only did the displacement suggest pneumothorax, but the bell sound and metallic tinkling were both heard, though, no doubt, both of them were produced in the stomach.

It might be supposed that the diagnosis might be made by finding irregular patches of dulness in different parts of the side; but, in the first place, the patients are generally too ill to permit of complete examination; secondly, the dulness of a solid organ like the spleen is easily marked by the general hyper-resonance of the intestines; and even if dulness be found, it may be due, as this case shows, to the presence of fluid, either blood or serum.

Abdominal Symptoms.—If life be prolonged, the symptoms may become chiefly abdominal; they depend upon the interference with the action of the stomach and intestines by the displacement. The patients may then complain of much dyspepsia, with pain and dyspnœa after food, and irregular vomiting; emaciation will follow, and the diagnosis of malignant disease be probably made.

The symptoms of internal strangulation sometimes develop, but this appears to be much less likely than might *a priori* be expected.

The period that may elapse from the time of injury to the development of symptoms is sometimes remarkably long.

Thus, in a case recorded by Peacock, though the symptoms developed only about three months before death, the injury to which the rupture must have been due occurred fifteen years before.

The interesting case which follows is recorded by Hale and Goodhart.

A gentleman, 49 years of age, was invalided home after an active life in India, having been losing health for two years. His chief symptoms were occasional vomiting and waterbrash. The vomit was often of large amount, and contained much dark-coloured mucus. In the later stages of his illness he lost flesh rapidly, and died of exhaustion after about three months' severe illness. He was thought to be suffering from cancer.

The *post-mortem* examination showed a hole in the diaphragm between the crura, extending from the œsophageal opening on the left to the vena cava on the right. Through this aperture a hernia had taken place, consisting of two-thirds of the stomach, a loop of the transverse colon, the lesser omentum, the greater part of the pancreas, and duodenum. The hernia was contained in a sac lying across the front of the spine, just above the diaphragm.

It was thought to be a congenital malformation of the diaphragm, which had rendered this a weak spot with subsequent bulging.

The patient had led an active life, and presented no symptoms until three years or so before his death.

Treatment.—There can be but one object in treatment, viz., to return the displaced organs into the abdomen and keep them there.

Unfortunately the correct diagnosis is the first essential, and this is generally not made.

Immediately after the injury, the collapsed condition of the patient, which renders examination difficult or impossible, puts operation out of the question. Even if the correct diagnosis were then made, the probable existence of other grave lesions would have to be taken into account.

I do not know of any instance in which a congenital case has been correctly diagnosed, and operation performed or even suggested.

It is stated that operation would be less difficult from above through the pleura than from below through the abdomen. It is difficult to see how this can be, for nothing short of an enormous opening into the chest would give such a

view of the diaphragm as would enable the operator to stitch up the rent, while the risk of laying the pleura so freely open under such conditions would in itself be very great.

75. HERNIA OF THE LUNG.

Hernia of the lung is a very rare condition. The cases fall into two groups —the traumatic, to which the great majority belong, and the non-traumatic, many of these being of congenital origin.

With penetrating wounds of the thorax, pneumothorax is the usual result, and the lung collapses; but in some instances the lung is forced into the wound, and protrudes through the chest walls. In course of time the protruding portion may slough off or be removed with the knife, and the condition heal without any evil effects following.

The diagnosis is easy, for the lung is visible.

In some instances, after the wound has healed externally, a hernia of the lung may develop beneath the scar, the protrusion taking place either through a weak, imperfectly healed spot, or through an opening in the thoracic walls which has remained unclosed.

It might be supposed that this would be a not altogether uncommon occurrence after incision for empyema, in which a portion of one or more ribs had been excised; but, as a matter of fact, it is very rare, and no instance has occurred within my own experience.

The rarest form of hernia of the lung is that which occurs without any evidence or history of trauma.

The hernia must, I suppose, in these cases be due to a weak spot or actual hole in the thoracic walls of congenital origin. In the cases recorded, the usual seat has been in the lower intercostal spaces (seventh, eighth, or ninth), about the junction of the cartilage with the rib; in fact, where the external intercostal muscles end.

The bulging of the lung, which is sometimes met with above the clavicle in emphysema, has been also described as hernia, but it hardly belongs to this group.

I have met with only two instances of this condition, both in middle-aged men; in the one a small tumour, the size of a filbert, was present on the second right intercostal space, about three inches from the sternum; the other, a somewhat larger tumour, was in the axilla, near Marshall's weak spot.

In a remarkable case recorded by Pietkiewitz there were two herniæ, both on the left side, and easily replaceable, the one between the sixth and seventh ribs in the nipple line, and the other behind, between the fourth and eighth ribs in the scapular line.

The swelling is soft, elastic, crepitant, resonant on percussion, diminishes or may disappear on inspiration, and becomes larger on expiration, especially if violent, as in coughing.

The hernia may be contained in a sac or pouch formed of dilated pleura. It is often replaceable, and may be easily kept back with a pad. The condition is interesting, but of no importance, and produces, as a rule, no symptoms.

No treatment is called for, but in the traumatic cases, if there seemed to be any indication, there would be no reason why the protruding portion of the lung should not be removed and the hole closed.

In the non-traumatic and congenital group, considering the risk of starting some infective process in the pleura, the condition would in most instances be wisely left alone.

76. SYPHILIS OF THE LUNGS AND PLEURA.

There is no reason why syphilis should spare the lungs and pleura, nor does it; yet visceral syphilis has its favourite seats, and the lung is not one of them. Even many of the recorded cases are not conclusive, for the diagnosis is, to say the least of it, open to question as being rather a matter of opinion than of demonstration. During life the diagnosis rests upon uncertain grounds, viz., first, upon the evidence or history of syphilis in the patient, and, secondly, upon the curative effects of iodide of potassium and mercury. Even after death the difficulties of diagnosis do not vanish, inasmuch as the lesions of syphilis closely resemble those of tubercle, for gummata and tubercular masses may be undistinguishable except by the discovery of the tubercle bacillus, and many of the fibroid indurations, which were once thought to be syphilitic, are now regarded as undoubtedly tubercular, or are referred to other causes.

Syphilis of the Pleura, except as a secondary affection, is practically unknown. There is, I believe, no pathological specimen extant in which undoubted syphilitic lesions existed in the pleura alone. The cases which have been diagnosed as syphilitic pleurisy during life are those in which pleurisy, whether with effusion or without, has developed in syphilitic persons, and been rapidly cured under the administration of iodide of potassium and mercury; but these are not sufficient grounds for such a diagnosis, which therefore rests on opinion only.

When the pleura is affected, it becomes involved, secondarily, by extension of inflammation from some syphilitic lesion in the neighbourhood. The commonest and most conclusive instance of the kind occurs in those cases in which there are syphilitic lesions of the chest walls, *e.g.*, periostitis, necrosis, or caries of the ribs.[1]

In a similar way, if gummata or sclerotic processes in the lung reach the surface, the pleura over the lesions becomes thickened and adherent, or it may possibly become actively inflamed, so that an effusion forms.

In either case the pleurisy which ensues is of a simple character, and typical syphilitic lesions are not found in the pleura.

Syphilis of the Lung.—The lesions in the lung which are referred to syphilis are—

1. *Pneumonia alba.*
2. *Gumma.*
3. *Fibroid induration.*

1. *Pneumonia alba—White hepatisation—Syphilitic pneumonia.*—All syphilitic lesions of the lung are essentially interstitial, and so it is with syphilitic pneumonia, which consists in the main of connective tissue hyperplasia, associated with proliferation and degeneration of the alveolar epithelium.

This condition is met with in the lungs of children who have either been born dead or have died shortly after birth. The change frequently involves a considerable extent of the lung, but it may occur in small isolated patches. Where the infants have breathed, some of the alveoli in the affected part may contain air, but most of them are filled with degenerated and desquamated epithelium. Whether the child have breathed or not, the lung in places may be found in a condition of arrested development, so that in the midst of the imperfectly developed connective tissue the bronchi appear in the embryonic state with gland-like epithelium.

[1] *Cf.* Nikulin's case, *Berl. kl. Woch.*, 1891, No. 40.

The affected parts contain little blood, and are pale or gray in hue, the term pneumonia alba being suggested by their lack of colour.

The condition just described is that which is usually meant by the term pneumonia alba, but by some authors it is applied also to another kind of change found in the lungs of syphilitic infants, in which, without interstitial change, the consolidation and gray colour are due to the accumulation in the alveoli of desquamated epithelium which has undergone fatty degeneration.

K. Fowler[1] refers to three cases of infants with congenital syphilis, associated with pulmonary lesions regarded as syphilitic, in which *post-mortem* examination proved the lesions to be of tubercular and not of syphilitic nature.

2. *Gummata*, also, are more frequently met with in the lungs of syphilitic infants than in those of adults. The gummata are nodules of granulation tissue which may undergo caseation in the centre. They are surrounded by a more or less definite capsule of fibrous tissue, from which processes may extend some distance into the neighbouring interstitial tissues of the lung.

They are usually few in number, but may be numerous. They may remain discrete or become confluent, and then produce masses of considerable size, *e.g.*, that of a pigeon's egg or Tangerine orange. Sometimes they are very small and peribronchial in position, resembling exactly the similar nodules found in chronic tuberculosis.

In the early stage they are grayish, red or white, semi-transparent, and of the size of a pea or even a nut; subsequently they become opaque in the centre and gray. They may, it is stated, undergo softening and break into a bronchus, and thus lead to the formation of a cavity.

It is clear that these lesions might often equally well be of tubercular nature. As many of the cases were described before the discovery of the tubercle-bacillus, or, if subsequently, without mention being made of any search for the bacillus, their real nature must remain doubtful.

Fig. 166.
Disseminated gummata in the lung. (From a preparation in the Museum of the R.C.S.)

The most satisfactory recent case was observed by M. Balzer, and is quoted by Lancereaux.[2] It occurred in a man of 32 years of age, who had numerous gummata in the liver, and had similar nodules in the lower part of the right lung and pleura. Examination was made for tubercle bacilli, but none were found.

[1] Clifford Allbutt, *Med.*, vol. v. 314.

[2] *L'union Médicale*, 1891, No. 13.

In others than infants, *i.e.*, in children of some years of age, and in adults especially, syphilitic changes of any kind are rare. In some instances, localised masses, regarded as gummata, have been met with; but for the most part the lesions described as syphilitic have been diffuse or localised fibroid changes in the course of acquired, and not of congenital, syphilis.

3. *Fibroid Induration.*—The fibroid induration may be either a diffuse, widespread change, involving a considerable portion of the lung, or may form localised nodules, which may reach a considerable size. This has been called *Syphilitic Fibrosis* of the lung.

According to some authors, localised pleuritic thickening, spreading for some distance along the trabeculæ of the lung or between the lobes, is syphilitic in nature; but such changes obviously have nothing characteristic about them, and may be, and doubtless often are, due to other causes.

Another form, which some authors regard as characteristic, is that in which the fibrous tissue spreads from the hilus some distance out into the lung, along the bronchi and main vessels. This condition is a very rare one, and in the cases, apparently similar, which I have seen, microscopical examination has proved the growth to be of a malignant nature (scirrhus), and not simple fibrous tissue.

It is obvious that, from the point of view of strict morbid anatomy, the subject of syphilis of the lung and pleura is in a very indefinite state. This was very evident in the discussion at the Pathological Society in the years 1876, 1877. Many of the cases then brought forward would probably not now be regarded as syphilitic at all. Nor of recent years has much advance been made, except to throw still further discredit upon the diagnosis.

As to the general frequency of syphilitic lesions of the lung, we must take the figures as they are provided for us, indefinite and unsatisfactory, possibly, as many of the cases are.

Haslund[1] records, that of 6000 syphilitic patients admitted into the hospital at Copenhagen during a period of seven years, 2 cases only were diagnosed as suffering from pulmonary syphilis during life.

The *post-mortem* records of the same period show, that out of 105 autopsies of children with congenital syphilis, undoubted lung-syphilis was found in 4 cases only; and that out of 18 autopsies of adults with acquired syphilis (8 males and 10 females), lung syphilis was found in 3 (1 male and 2 females). In only 1 of all these 7 cases was the condition diagnosed during life.

Heller[2] states that, of syphilitic children, 50 per cent. show interstitial pneumonia, and more than half these have pneumonia alba, while of non-syphilitic children only 20 per cent. show lung affection of any kind. It is only in still-born children that the most marked forms of pneumonia alba are found.

Hiller[3] analyses 87 cases, out of which, however, he rejects 29 as being either doubtful or combined with tuberculosis. He found the lesions in the following proportions:—

1. Peribronchial, that is to say, connective tissue growths round the small bronchi, in 21 cases.
2. Circumscribed peribronchial nodes in 8.
3. Diffuse lobular indurations in 14.
4. Gummata in 9.

In 2 cases he described a proliferating lymphangitis, which he regarded as of a specific nature.

After a careful analysis of the various lesions, he concludes that with a more persistent and careful examination for the tubercle bacillus, it is quite likely that syphilitic disease of the lung will ultimately disappear from literature. Without, perhaps, going quite so far as this, we may agree with him that the subject is in an extremely indefinite condition, and that, as Fagge stated some years ago, the subject requires to be investigated anew by unbiassed observers.

K. Fowler states that there are only 12 instances of syphilitic disease of the lung in adults in the museums of all the London hospitals together. In most of these, though the question of tuberculosis was carefully considered, the absence of bacilli does not appear to have been proved.

[1] Hosp. Tid., 3 R., viii., Nos. 15 and 16, 1890. Abstract, *Virch. Jbt.*, 1890, ii. 631.
[2] *Deutsch A. f. kl. Med.*, xlii. 1887.
[3] *Virch. Jbt.*, 1884, ii. 543.

Physical Signs and Symptoms.—Considering that the diagnosis of the disease is so much a question of morbid anatomy, it may seem unnecessary to refer to the physical signs and symptoms of the disease, which, from the nature of things, will present little or nothing characteristic.

The infants are still-born, or die as syphilitic children do soon after birth, and usually present no definite physical signs.

In some few cases, pulmonary lesions, which have been referred to congenital syphilis, have been found late in life, one case even at the age of 40 years, an observation,[1] if correct, unique.

In adults, in most cases, the disease is the result of acquired syphilis, and belongs to the tertiary stage of the disease, as do the other forms of visceral syphilis.

Dieulafoy,[2] who has written one of the most recent papers on this subject, makes five clinical types of the disease—

1. That which resembles tubercular broncho-pneumonia.
2. That which resembles tubercular phthisis.
   Both of these are diagnosed by the results of anti-syphilitic treatment only.
3. Broncho-pulmonary sclerosis with pleuritic thickening.
   Diagnosed only by the appearance of other syphilitic signs.
4. Syphilitic and tubercular changes combined.
   a, syphilis primary ; *b*, tubercle primary.
5. Hereditary syphilis of the lung, early and late.

Yet he concludes that in affections of the chest it is more prudent to think of syphilis, and to be on the lookout for it, rather than to diagnose it during life.

For the most part, syphilitic disease leads to chronic indurative lesions, the symptoms and physical signs of which resemble those of fibroid phthisis or other fibroid disease of the lung. They are stated, however, to be, as a rule, unilateral, and to affect the middle or lower portions of the lung, and in this respect to more closely resemble chronic interstitial pneumonia or chronic pleurisy.

If the lesion be localised and not of large extent, there may be no symptoms at all ; but if widespread, it is attended with shortness of breath. If it should involve the root of the lung, the lesion is likely to compress the air-tubes and vessels, and the signs would resemble those produced by a malignant growth starting in this position.

Syphilitic Phthisis.—In some cases it has been supposed that syphilis may lead to symptoms resembling those of phthisis. This may be presumed to occur where the syphilitic lesions, gummata especially, have broken down in the centre, and, after communication with the bronchus, been expectorated, a cavity being in this way formed which may subsequently extend. These cases have sometimes been described as syphilis of the broncho-pneumonic or phthisical type, and the cases have been denominated syphilitic phthisis.

Syphilitic phthisis is a relic of the time before the unity of phthisis was established. Miners' phthisis, grinders' phthisis, diabetic phthisis, and others, which were once regarded as special forms of phthisis, are now proved, by the presence of the tubercle-bacillus, to be tubercular disease of the lung occurring in miners, grinders, and diabetics respectively ; and so, no doubt, will syphilitic phthisis prove to be merely tubercular disease in a syphilitic person.

I do not think there is any undoubted case recorded of progressive destructive disease of the lung in a syphilitic patient which has proved to be of non-tubercular origin. Until such cases are conclusively demonstrated, we must regard the existence of such a disease as syphilitic phthisis as extremely doubtful.

[1] Lancereaux, *Union médic.*, 1891, No. 13.

[2] *Gaz. hebdom.*, 1889.

There is a group of cases in which non-tubercular destructive lesions have been found in the lung in connection with syphilitic stenosis of the trachea or bronchi. Careful examination of these cases leaves little doubt, I think, that these destructive lesions are not syphilitic, but of the same kind as those which follows stenosis of the air-tubes of other, *i.e.*, non-syphilitic, origin, *e.g.*, that due to a new-growth, an aneurysm or an enlarged gland.

But for all that there may be a relation between tubercle and syphilis. Syphilis may predispose to tubercle, just as any other cachexia may, so that syphilitic patients may actually be more liable to acquire phthisis than other patients. Yet I do not know that even this is true, for there are no figures to show that phthisis is more prevalent among syphilitic patients than among the non-syphilitic; and I cannot help thinking that if it were a fact, statistics to prove it would have been forthcoming.

Although syphilis cannot, as far as we know, produce a progressive destructive disease of the lung, still there is reason to believe that it may produce fibroid induration of the lung; the question would then arise, How far can we distinguish the fibroid lesions due to syphilis from those which are due to tubercle and other causes? It is suggested, in the first place, that they are, for the most part, unilateral, and affect the lower parts of the lung; but this would not serve to distinguish syphilitic disease of the lung from other forms of interstitial pneumonia. Besides, we know that tubercle may begin in the base, though its usual seat is the apex, while there are cases recorded in which affections of the lung referred to syphilis have been found at the apex, so that localisation is not much assistance in the diagnosis.

The history and evidence of syphilis in the patient are, again, no sure guides, for the general liability of syphilitic patients to phthisis is not known.

The point upon which chief stress is usually laid in the diagnosis of syphilis of the lung is the effect of treatment; that is to say, the rapid curative effects of iodide of potassium and mercury. This, however, is almost as inconclusive as any of the others, for mercury and iodide of potassium have been used, and with benefit, in tuberculosis of the lung.

As to the existence, then, of syphilitic phthisis, that is to say, of progressive destructive disease of the lung produced by syphilis as distinct from tubercle, we have no satisfactory evidence. The fibroid lesions, referred to syphilis, and which have been called *syphilitic fibroid phthisis*, cannot be distinguished during life from fibroid induration of tubercular or other origin. Pathological evidence alone is conclusive, and that in many cases is also defective and uncertain.

It would be wise, therefore, completely to discard the use of such a term as syphilitic phthisis, and when the facts seem to justify the diagnosis, to describe the condition simply as syphilis of the lung.

77. PNEUMONO-MYCOSES.

Micro-organisms are provisionally arranged by Pfluger into four groups—

1. *Hypho-mycetæ* or mycelium-fungi.
2. *Blasto-mycetæ* or yeast fungi.
3. *Streptothrix.*
4. *Schizo-mycetæ* or fission fungi.

The schizo-mycetæ or bacteria form the most important pathogenic group, for it includes those organisms to which some of the gravest pulmonary affections are due, viz., tuberculosis, pneumonia, anthrax and glanders. These diseases have already been fully dealt with.

The other groups contain but few pathogenic forms, that is to say, they rarely produce pathological lesions themselves, but may be found occasionally in the lungs, associated with other lesions as the result of accidental infection. Thus the oidium albicans, so common in the mouth, has been also found in the bronchi, and even in the lung-tissue. The group of streptothrix contains one very important pathogenic organism, the actino-myces, and possibly one or two other allied forms.

Of the hypho-mycetæ many have been proved by experiment to be pathogenic in animals, but none of them appear to be met with in man, except the aspergillus fumigatus, and possibly, also, the aspergillus niger.

Lastly, of the lower forms of animal life, a few instances of protozoic infection have been recorded under the name of *coccidiosis*.

Actino-mycosis and aspergillosis are practically the only two affections which deserve more than a passing reference.

78. ACTINOMYCOSIS OF THE LUNG AND PLEURA.

Actinomycosis is a disease due to the growth of a peculiar fungus, with the consequent reaction and degenerative changes in the tissues involved.

Fig. 167.

Actinomycosis, showing mycelium with sporelike bodies between the filaments from the pus removed from the pleural cavity. (Delepine, *Path. Soc. Tr.*, xl. p. 431.)

Fig. 168.

Actinomyces from a liver abscess in the same case, showing clubs. (Delepine, *Path. Soc. Tr.*, xl. p. 431.)

The fungus grows in a peculiar way, in clumps or clusters, the central portion of which is formed of a dense mycelium, and the peripheral part of peculiar club-shaped masses arranged in radiate fashion, to which it owes the name actino-myces or ray fungus.

The clumps are visible to the naked eye, and can be detected in the pus, or in the tissues, as minute yellowish granules. If these be picked out, placed upon a slide and examined, without pressure from the cover glass, the appearance is presented which is shown in the figure. On teasing the specimen and staining it by Gram's method, the mycelium is stained blue and the clubs pink. It is then seen that the mycelium is composed of a central thread-like core, which stains blue, and an outer sheath which stains pink. The clubs are formed of the swollen sheaths of the mycelium, and the central core terminates in the club, either as a fine filament or network of filaments, or with an expanded end. In some parts the mycelium is somewhat swollen, and contains small round masses, which stain with difficulty; these are probably spores.

The fungus has been cultivated, and yields much the same appearance, but it does not, under ordinary circumstances, form clubs. By some authorities these clubs are regarded merely as degenerative forms, and not as a necessary part of the development of the fungus.

The spores, it is stated, sprout into extremely fine, straight, sinuous, or sometimes spiriliform threads, which branch irregularly, but sometimes dichotomously, to form the mycelium, from which the clubs develop. It is probable that the propagation within the body is by means of the wandering cells, which carry the spores away to a distance, and there set up fresh centres of growth.

PATHOLOGY.—The changes produced in the tissues consist primarily of nodules of granulation-tissue, which, by confluence, may form tumours of considerable size, and then resemble the granulation tumours, *e.g.*, sarcoma, lympho-sarcoma, by which names they have been often described.

Closely associated with the formation of granulation-tissue is the development of connective tissue, which tends to encapsulate the fungus. Where many nodules are confluent, a coarse, irregular meshwork of connective tissue may be produced.

The next change is a central necrosis, probably associated with suppuration. The cells in the centre in the neighbourhood of the fungus necrose, and may then present a caseous appearance. If they become the seat of suppuration, small abscesses are formed which gradually increase in size, and communicate one with the other. The section of a tumour in such a condition would present the appearance of a sarcoma-like mass, intersected with bands of connective tissue and riddled with small pus-containing cavities or tracts.

In the tissues immediately surrounding such tumours, fresh nodules of fungus growth may be found, resembling in this respect, very closely, the miliary tubercles not infrequently met with round a chronic tubercular mass.

The changes are of very slow development, so that the tumour may take months to reach any great size so long as no suppuration occurs in it, but when suppuration takes place, the extension is usually rapid.

Extension of the growth takes place chiefly by contiguity, new nodules developing in the neighbourhood of the old ones, and ultimately coalescing with them. The lymphatic glands are, as a rule, not involved, as they are in tuberculosis or inflammatory processes, so that the disease may remain for a long time purely local. It is even possible in such cases that cicatrisation may gain the upper hand, and thus in the end the fungus become encapsulated, or even destroyed. Under such circumstances, at the last nothing may be left but a mass of fibrous tissue, in which all trace of the original cause may be lost.

When suppuration occurs in the new tissues, the progress is usually more rapid, and the question has been much discussed, whether suppuration is caused actually by the fungus itself or by some secondary infection with pyogenic organisms. It is probable that the fungus is itself capable of exciting suppuration, for if the suppuration were due to secondary infection, we should expect to find the lymphatics affected, and they usually escape; while in some of the cases which have been examined bacteriologically for the purpose, no pyogenic organisms have been found; but, of course, secondary infection with pyogenic

organisms may occur, especially when the growth has reached the external parts of the body.

Occasionally metastases occur, and secondary growths develop in many parts of the body. This is, in some cases, the result of the fungus having made its way into a blood vessel, vein, or artery, as the case may be; the metastatic growths pass through the same changes as have been already described, though it appears that suppuration is somewhat more frequent. When actinomycotic abscesses are thus found in different parts of the body, the cases are often described as the "*pyæmic form*" of actinomycosis. The secondary growth may then be found in almost every part of the body, *e.g.*, lung, spleen, kidneys, liver, brain, muscles, subcutaneous tissue, and even in the heart itself.

The history of the ray fungus outside the body is not known, but it probably gains access to the body chiefly through the mouth, in connection with some article of food, possibly corn.

In cattle.—In cattle the lesions of actino-mycosis have long been known, though not referred to their proper cause. They have been described under the names of wens, clyers, tuberculous abscesses, polypi, lymphoma, cancer of the tongue, lumpy jaw, bone cancer, bone tubercle, osteo-sarcoma and fibroplastic degeneration of bone.

In cattle the skin and subcutaneous tissues are the favourite seats of the disease, producing the so-called wens or clyers; so also are the upper and lower jaws. In the mouth, tongue, and pharynx the disease is not so common.

Of the respiratory organs the lungs and pleura are perhaps the commonest seat, and here the appearances are like tubercle, with which the disease has been most commonly confused.

In the larynx, trachea, and bronchi also, polypoid growths, either sessile or pedunculated, have been found.

In man.—Its common seat in man, as in animals, is the mouth, chiefly the jaw, probably as the result of infection through a carious tooth. Many cases are described also in the intestinal tract or abdominal viscera, and in a few cases the primary seat of infection has been in the skin.

The general frequency of pulmonary actino-mycosis in man may be estimated from the figures given by Rutimeyer,[1] who found that out of 105 published cases of actinomycosis, 22 were primary in the lung or pleura.

Sabrazès and Cabanès[2] state the relative frequency of the pulmonary form to be 12–15 per cent. Still the frequency is probably greater than the figures show, for attention has but recently been drawn to the disease, and many cases have no doubt been overlooked or wrongly attributed to tubercle, to which disease actinomycosis of the respiratory organs presents so many clinical and pathological resemblances.

The *larynx* and *trachea* might be the seat of primary growth in man, as they are occasionally in cattle; but there is, so far as I know, no case of the kind recorded. They might also become secondarily involved by extension from the pharynx, œosophagus or neck.

The *bronchi* might be primarily involved, but there is only one case of the kind recorded, viz., that of Canali, and in this the diagnosis is not altogether satisfactory, because there had been no *post-mortem* investigation.[3]

[1] *Berl. klin. Woch.*, Jan. 21, 1889, p. 45.
[2] *Rev. de Méd.*, Jan. 10, 1899.
[3] *Cf.* J. Israel, *A. de Menschen*, 1885.

This case occurred in a girl of 15, who, for eight years, had been suffering with cough and expectoration. The sputum was scanty, viscid, fœtid, and contained small, yellow, greenish lumps, which proved, on examination, to be actinomycotic.

The patient was improved by treatment, but not cured, and was living at the time that the description of the case was published.

The case was called by Canali Bronchitis actinomycotica, but in the absence of a *post-mortem* examination the diagnosis must remain doubtful.

The *lung* may become involved either primarily or secondarily.

Where it is affected secondarily, the disease may reach it either by direct extension from a growth in its neighbourhood or by metastasis.

Where the lung is involved by direct extension from other parts, the primary growth must be seated in its neighbourhood, *e.g.*, in the mediastinum or œsophagus, or it may spread through the diaphragm from the abdomen.

When metastasis occurs, the infection is generally carried through the blood vessels, and the lesions in the lung are associated with metastatic growths in other parts. But the lung may infect itself by the infected sputum being sucked into the air-tubes, and starting fresh foci of inflammation and growth. In some cases this may resemble acute tuberculosis very closely.

Where the growth in the lung is primary, the fungus will have gained access by the air-tubes, and been carried by the air into the small bronchi, or possibly into the ultimate vesicles of the lung. In these cases the original source of infection may have been in the mouth, as is illustrated by a case of Israel's,[1] in which, in an actinomycotic cavity in the lung, a portion of tooth was found.

When the fungus has thus reached the small bronchi or bronchioles, inflammation is set up, and small broncho-pneumonic patches develop, forming the nodules, which in the centre show, quite early in the disease, a yellowish white colour, due to the necrosis of the cells surrounding the fungus.

Fig. 169.

Actinomycosis of the lung (*Ziegler*). Round the fungus, which is diagrammatically represented in the centre, is a small-celled infiltration with dilated vessels. The vesicles outside this nodule are in a condition of early pneumonic consolidation.

The subsequent changes set up in the lung, by the fungus, are those of a subacute inflammation.

The morbid tissue-changes are best described in connection with the accompanying figure. Round the fungus develops a mass of cellular inflammation, in which may ultimately be found both epithelial and giant cells. Round these nodules an abundant formation of connective tissue takes place. The vesicles in the neighbourhood become filled with catarrhal, epithelial and small white cells, and ultimately they become completely obliterated by the growth of vascular connective tissue from the septa into their cavity.

[1] Israel, *Arch. f. klin. Chir.*, 1886, xxiv. p. 160.

By the development of fresh nodules round, and their confluence with the original ones, a considerable mass may be formed, and the greater part, or the whole, of a lobe become involved.

The longer the case lasts, the more connective tissue will be developed, and so a considerable and dense fibroid induration of the affected parts be produced.

It is even conceivable that the connective tissue may completely isolate the fungus and destroy it, so that in the end nothing is left but a fibrous mass, in which all trace of its origin may be lost. In some cases the amount of fibrous tissue is out of all proportion to the amount of actinomycotic growth, so that the fungus may be very difficult to find.

Usually the inflammatory nodules surrounding the fungus undergo central necrosis and softening, and thus irregular cavities form which contain fatty detritus, fatty cells, pus cells, some red blood cells, some fluid, and the fungus.

Section of such a portion of the lung presents a mass of fibroid induration, riddled with tracts and cavities containing pus; and in the surrounding tissue small grayish or yellow points or granules may be seen, which prove to be new fungus growths. These lesions closely resemble those of chronic tuberculosis. The diagnosis would, however, be easily made by the discovery of the minute granules of fungus in the pus.

The inflammation in the lung involves the pleura, and leads to the adhesion of its surfaces and to the more or less extensive obliteration of its cavity. Through the adhesions the process may extend to the parts adjacent; thus it may reach the pericardium or even the heart itself, the mediastinum, and, by passing through the diaphragm, the organs of the abdomen or the peritoneum. More frequently it spreads to the chest walls, and as soon as it penetrates them, leads to superficial fluctuating swellings in the subcutaneous tissue, which closely resemble a pointing empyema; and it is with this that most of the cases are at first confused.

When the process has thus reached the superficial tissue, it spreads rapidly, so that in a few weeks a very extensive area of the chest walls may be affected; the abscesses burst or are opened, and leave fistulous tracts of an unhealthy, granulating appearance, discharging a small amount of thin pus, in which the actinomyces may be found abundantly.

The secondary lesions outside the lung may often reach an extent which is extraordinary compared with the amount of primary mischief in the lung itself; indeed, this may even cease to extend or may cicatrize, so that the primary origin in the lung might be easy to overlook.

In the course of the case, *general infection* of the blood may occur. This is due to the direct breaking of the actinomycotic growth into some vessel, either a vein or an artery, and then metastatic growths may develop in almost every part of the body. Some of these may continue as solid tumours, and resemble, in some respects, infarcts, from which, however, they are distinguished by their shape and situation. Others suppurate and form abscesses, so that the case comes to resemble one of pyæmia. Such cases are often described as the *pyæmic form* of actinomycosis, and of this a fair number of cases are recorded, especially in the course of primary pulmonary actinomycosis; it would seem to be really more common in this than in other forms.

The metastasic growths may be found in all organs of the body—in the spleen, liver, kidneys, lungs, brain, in the general muscular subcutaneous tissue, and even in the substance of the heart itself.

Primary Actinomycosis of the Lung.—In *primary* actinomycosis of the lung, the favourite seat of the initial growth is in the lower lobe, and this stands in strong contrast to tubercle, in which the initial seat of lesion is usually the apex.

An interesting case of primary lung actinomycosis, in which both apices were involved, is recorded in the *Clbl. f. klin. Med.*, vol. xlviii.

Secondary Actinomycosis of the Lung.—When the actinomycosis of the lung is *secondary*, its distribution is, of course, more irregular.

Secondary actinomycosis of the lung may arise in two ways—either by direct extension from parts adjacent or by metastasis.

1. *In the case of direct extension*, the original seat of the growth is often in the œsophagus, whence it spreads to the tissues of the neck or mediastinum. The trachea may thus become involved directly, and the growth, penetrating to it, may be sucked into the air-tubes and thus infect the lungs. I do not know that there are any instances in man in which either pressure upon the trachea, or stenosis consequent on the infiltration of its walls, has been produced; nor have any cases, so far as I know, of polyp in the trachea been recorded, a lesion which is not uncommon in cattle.

Usually, when the tissues of the neck become involved, the growth spreads downwards, following the course of the cervical fascia, so as to reach the mediastinum, whence it spreads to the pleura and lung.

In other cases the lower part of the œsophagus is the primary seat of lesion, and then the mediastinum becomes involved early, or possibly one of the main bronchi. The involving of the lung may then come about in two ways—either by direct extension, or by the growth bursting into the air-tubes and being aspirated into the lung.

No doubt it is through the œsophagus that the cases of apparently primary vertebral actinomycosis arise.

Of course it is possible for the thoracic viscera to be involved secondarily to primary actinomycosis of the walls of the thorax, but I do not know that any case of this is recorded; usually it is the other way, the viscera being affected first and the chest walls subsequently.

When the primary actinomycosis is in the abdomen, as, for instance, in the liver, spleen, or peritoneum, the process may spread upwards, involve the diaphragm, and so reach the pleura and lung.

2. *In the case of metastasis*, extension rarely takes place through the lymphatics even when suppuration has occurred, and this is a strong argument in favour of the suppuration being the direct result of the actinomycosis itself, and not due to secondary infection by pyogenic organisms. Metastasis usually seems to take place through the blood vessels, owing to the growth having penetrated one of them, and this has been actually demonstrated in one or two instances.

Even in some cases of apparently primary actinomycosis of the lung, the disease may be really secondary, and have its origin in the neck or mediastinum, and occasionally even in the mouth, as in Israel's case, already referred to.[1]

The lung may even infect itself; that is to say, the primary growth in the lung may make its way into a bronchus, and thus the infective sputum be sucked into the other air-tubes and start fresh foci of disease; or if the growth should penetrate the pulmonary artery, it may be carried all over the lung, and produce a sort of general miliary actinomycosis of the lung. The former process,

[1] Israel, *l.c.*

which may be described as inhalation-infection, explains what is not uncommonly seen round these actinomycotic growths, viz., numerous foci of recent growth surrounding an older mass, just as recent tubercles are often found round old tubercular lesions.

Analysis of Thirty Cases.

I have collected[1] 30 cases of primary actinomycosis of the lung from published records, in 25 of which there is an account of the *post-mortem* examination.

Sex.—The sex is specified in 27, 17 males and 10 females; the sexes being affected, therefore, in the proportion of about 3 males to 2 females.

Age.—The age is given in the following table:—

Up to 20	4	M. 6; M. 9; M. 19; M. 20.
20 to 30	9	M. 21; F. 22; F. 24; M. 28; F. 28; (3) M. 30; F. 30.
30 to 40	6	F. 31; M. 34; M. 37; (2) F. 39; F. 40.
40 to 50	3	M. 45; F. 50; M. 50.
50 to 60	1	M. 52.
Above 60	1	M. 63.
Total,	24	

The two youngest cases were children of 6 and 9 years of age, and the oldest a man of 63.

Side affected.—Of 27 cases, the left side was affected in 17, the right in 8, and both in 2. The left lung is, therefore, more liable than the right in the proportion of at least 2 to 1.

Seat.—The part of the lung affected is commonly the lower lobe.

This was involved 13 times, as compared with the upper 3 times, and the middle 4 times. By far the favourite locality of primary disease is the lower lobe of the left lung.

Where both lungs were affected, in one both bases were the seat of disease, and in the other the lower lobe of the right lung and the upper lobe of the left.

The Result.—Of the 30 cases, one was discharged *in statu quo*, and the ultimate result is not recorded; one was stated to have been cured with Iodide of Potassium, and one to have been much improved under the use of injections of Iodoform; the rest died.

Of the 30 cases, a full account of the *post-mortem* examination is given in 25. It is upon this that the previous account of the pathology of the disease is based; but there are a few other points of interest and importance which deserve to be mentioned here.

In no case, at the time of death, was the disease confined to the lung or pleura, but had extended to the parts around.

In nearly every case *the chest walls* had become involved, and superficial abscesses had formed, which had either been opened or had discharged spontaneously, and that frequently, in several places.

In most of these cases the first swelling made its appearance in the axilla, occasionally in the mammary region, and once or twice behind. It was rare for these swellings to appear in the upper parts of the chest; in one or two cases the swelling appeared above, near the sternum; but this was due to the fact that it was the upper part of the lung which had been the seat of original disease.

[1] *Path. Soc. Trans.*, 1897, vol. xlviii.

In 3 other cases, not included in this table, the lung, though considerably involved, was not the seat of primary disease, but had been involved secondarily, the primary disease having started in the mediastinum or in the parts around the vertebræ, the mischief having probably commenced in connection with the œsophagus.

The *pericardium* was involved in 7 cases, and was usually itself the seat of actinomycosis. In 2 of these cases there was apparently nothing more than a simple inflammation, with some effusion and adhesion. In the rest it was considerably thickened and indurated; and the new tissue contained many actinomycotic abscesses, and in 2 instances the process had spread to the muscular substance of the heart itself.

Just as the pericardium may be the seat of a simple inflammation, so may *the opposite pleura*, and in 2 or 3 of the recorded cases the opposite pleura was either adherent or contained fluid, serous in some cases and purulent in others. In one case there was a local empyema, containing 5 oz. of pus, in which no actinomyces could be found.

In 3 cases the disease involved the *muscles of the back*; in 2 of them spreading downwards between the pillars of the diaphragm, eroding many of the lower dorsal and upper lumbar vertebræ, forming abscesses in the psoas and quadratus lumborum muscles.

In one case, in which the disease spread upwards, it surrounded the jugular vein and caused thrombosis in it; in another an abscess formed in the walls of the superior vena cava, but did not perforate the vessel. In another case the disease involved the upper intercostal nerves and some of the branches of the lower cervical plexus, thus causing much neuralgic pain in the arm.

Amyloid disease was present in 3 cases, and, curiously enough, not necessarily in those of longest duration, or with the largest amount of suppuration.

Thus, in a man of 30, the disease had lasted six or eight months only, and there had been little suppuration; in a man of 37, although there had been a considerable amount of suppuration, the disease had lasted only six months; and, lastly, in a woman of 24, the disease had lasted about seven months, but there had been only a small amount of pus discharged through a small sinus for about five months.

Actinomycosis is not characterised by profuse suppuration as a rule, so that there must be some other explanation of the occurrence of amyloid disease, and I think it must be attributed to the disease itself.

In some cases the stress of the disease falls upon *the pleura* rather than upon the lung. In one instance the pleura was in a very unusual and curious condition; the lung was but little affected and was collapsed; the pleural cavity, on the other hand, was filled with a spongy, vascular, sarcoma-like tissue, soft and friable, in which actinomycotic abscesses were found.

A condition very similar to this occurred in a case of my own, of which an account is given at the end of this article.

Miliary dissemination of Actinomycosis in the Lung.—Of this I have met with 3 examples, though 2 of them are not included in this series of 30, because the primary growth appears to have been external to the lung. In one of these [1] the primary disease was in the liver; the lung was filled with actinomycotic bronchopneumonic patches of small size, but there was nothing to show how the fungus

[1] *Path. Soc. Trans.*, xlv. p. 237.

had gained access to the air-tubes. In another case the œsophagus was the primary seat of growth, whence the disease had spread to the mediastinum and to the parts near, bursting into one of the large bronchi as well as externally.

Usually this dissemination in the lung affects the lung on the diseased side only, but in one case the opposite lung and pleura were similarly affected as well.

The lesions look, in the early stages when they are small, very like acute miliary tuberculosis, with which, no doubt, they have been often confounded.

Metastasis.—In the 30 cases, there are 3 of the so-called *pyæmic form*, and 2 others in which there were secondary growths, which could not have arisen by direct extension. In one of the latter cases there were abscesses in the liver and spleen, but as both lower lobes of the lung were involved, the extension might have been direct to these organs. There were, however, together with these, in one case an abscess in the brain, and in the other case many independent abscesses in the kidneys.

The following is an account of the three pyæmic cases :—

Case 1.[1]—A female, aged 24 ; duration of the disease, seven months. Left lower lobe involved, pleura and diaphragm adherent ; the disease had spread along the diaphragm to the retroperitoneal tissue, and involved the psoas and quadratus lumborum muscles. Secondary abscesses were found in the right lung, which might possibly, however, have been due to inhalation. In the left kidney and liver were independent abscesses, and there was amyloid disease of the spleen.

Case 2.[2]—Female, aged 39 ; duration of the disease, eleven months. Left lower lobe involved, communicating with the abscesses in the chest walls. An abscess lay between the left lower lobe of the liver and the diaphragm, and there was serous effusion into the peritoneum. Numerous abscesses were found in the spleen and in both kidneys, and many soft tumours in the skin and muscles, all containing actinomyces. Besides this, the portal vein was filled with pus, which contained actinomyces, and there were numerous actinomycotic abscesses in the liver. The hepatic condition might be described as pylephlebitis actinomycotica.

Case 3.[3]—A male, the disease of some months' duration. Both lungs were affected in their lower lobes. There was a large abscess in the left lobe of the liver ; but besides this, many other abscesses in both kidneys as well as one also in the brain.

In all these cases, it will be observed, the secondary abscesses contained actinomyces, and the cases were not pyæmic in the strict sense of the term, *i.e.*, were not due to a secondary infection with pyogenic organisms, but were the result of the general dissemination of actinomyces.

CLINICAL HISTORY AND COURSE.—The onset of actinomycosis is generally indefinite, for it appears that so long as the disease is confined to the lung, it produces few or no symptoms ; it is not until the disease has made its way into the bronchus on the one side, or has reached the pleura on the other, that symptoms arise.

In the former case cough and expectoration develop, and the sputum may be found to contain the fungus.

When the disease reaches the pleura, pain is experienced ; either a constant, dull, aching pain, or else the sharp, stabbing pain of acute pleurisy. Occasionally the apparent onset of the disease is by an acute feverish attack, which may be of sudden onset and short duration. It may terminate after a few days' illness suddenly, just like pneumonia ; but in a short time, it may be after a few days only, it is likely to recur. In many of the recorded cases the subsequent illness is dated from attacks of this kind.

If there be any physical signs, they point rather to pleurisy than to pneumonia, and the case is usually diagnosed at this time as an empyema. Exploratory

[1] J. Israel, *l.c.* [2] *Ibid.*
[3] Mallory, *Boston Med. and Surg. Journal*, 1895, p. 297.

puncture, however, very often leads to no result, nothing at all, perhaps, being obtained; the needle appearing not to reach a cavity, but to be held fast in solid tissue, or, if pus be reached, a small quantity only is found, and that with difficulty, perhaps after several attempts.

Later, when an external fluctuating swelling appears, the diagnosis of empyema seems to be confirmed; but here, again, puncture may obtain but little pus; even on incision little may be obtained, although the extent of dulness would appear to indicate a large effusion. If an incision be made and the finger introduced into the pleura, it seems to reach tissue on all sides of it, and no large cavity is found. The condition then found, even if there be nothing previously to suggest the diagnosis, would no doubt raise the suspicion of actinomycosis, and the fungus would be easily found in the pus when looked for.

The physical signs are usually those of chronic pleurisy. There is considerable dulness at the base of the lung, and diminution of the voice- and breath-sounds; but at the same time usually a considerable retraction of the affected side. In one or two cases the voice- and breath-sounds have been increased, and there has been much crepitation, or even signs which indicated the existence of cavities. The physical signs, in conjunction with the history, have then led to the diagnosis of chronic interstitial pneumonia with bronchiectasis. If the apex were the seat of mischief, the resemblance to phthisis would be very close, and the diagnosis of actinomycosis could only be made by the discovery of the fungus.

There are generally well-marked **constitutional symptoms,** especially in the later stages; for instance, loss of flesh and strength, with acceleration of the pulse and respiration, and a rise of temperature. Indeed, in the condition in which most of the recorded cases come under observation, there is generally a well-marked hectic, the temperature reaching 103° or 104° every night.

It is probable that the fever is to be associated with the suppuration, but, as stated, the suppuration need not be due to infection with pyogenic organisms.

The sputum is generally nummular, mucopurulent, viscid, with the yellowish granules or streaks of actinomycotic growth. It does not contain elastic tissue or tubercle bacilli. It may contain blood mixed with the sputum, and be rusty or red in colour, thus resembling more the sputum of pneumonia than that of phthisis.

As in phthisis, the patients usually remain for some time capable of work; and though gradually losing strength and flesh, are not invalided except for the slight intercurrent attacks of fever or pleurisy. The more acute, pneumonia-like attacks which have been referred to are not very common.

The development of symptoms is, for a long time, slow; but as soon as the disease reaches the walls of the chest, the extension is rapid, and widespread suppuration may occur within a few weeks.

DURATION.—The total duration of the disease is short; a few months, or, at any rate, not more than twelve, bring most of the cases to an end, the disease being dated from the onset of acute symptoms; but it is really impossible to say how long the disease may have actually existed, because it gives no signs by which it can be recognised in the early stage.

Of 18 cases in which the probable duration was stated, it was from five to nine months in 9, from nine to twelve in 2, from twelve to eighteen in 7, from eighteen to twenty in 2, and 1 case possibly lasted three years. The shortest duration known is five months, so that 15 out of the 18 cases were fatal within eighteen months from the earliest onset of symptoms.

The Cause of Death.—In most cases the cause of death is gradual exhaustion, the patients dying much as they do with phthisis, malignant disease, or uncontrollable and prolonged suppuration. Many of the final symptoms depend upon the direction in which the disease has extended, and the organs which have thus become involved; thus, if the pericardium or heart be involved, the case may end with grave cardiac symptoms; or if secondary growths form in special places, as, for instance, in the brain, accidental and unusual symptoms may appear.

Where general dissemination occurs, that is to say, in the pyæmic forms, the symptoms added are those of a general pyæmia, and the patients usually die in that condition.

The most marked examples of this occur in those cases in which abscesses form in various parts of the body, for instance, in connection with the superficial parts, viz., in the subcutaneous tissue or muscles.

In the liver, spleen and kidneys, secondary growths may develop without marked signs, and the lesions may only be found *post-mortem*, without having given evidence of their presence during life.

When the dissemination is widespread in the lung, the symptoms resemble those of acute miliary tuberculosis.

THE DIAGNOSIS.—The diagnosis is easy enough, if there be the characteristic sputum or pus, but if not, it will be difficult or impossible.

The general symptoms, *e.g.*, loss of flesh and fever, suggest phthisis, but the localisation may be of assistance in the diagnosis from phthisis, since actinomycosis usually affects the base, and tuberculosis the apex; still, this is not conclusive, for there are instances of apex-actinomycosis, just as there are instances of base-tuberculosis.

On the whole, it is from empyema or some form of chronic pleurisy that the diagnosis has chiefly to be made. The signs are those of pleurisy and the localisation the same. Indeed, there is actually pleurisy and generally suppuration too, so that the diagnosis is so far correct; but it is as to the cause of the pleurisy that the difficulty arises, and this cannot be determined unless the characteristic fungus be obtained.

In children the disease might resemble chronic interstitial pneumonia with bronchiectasis; but these conditions are not common in the adult, while actinomycosis is rare in children.

Malignant disease has been diagnosed in some instances, and notably in the historical case of Lebert, and malignant the disease is in respect of its results; but the common forms of malignant disease are but rarely associated with hectic temperature. Still, this again is not an absolute rule, for inflammatory conditions, both in the lung and pleura, may arise in its course.

Again, no doubt, the pyæmic form has often been called pyæmia, from which it can hardly be distinguished, except by discovering the cause.

PROGNOSIS.—Of prognosis there is really but little to say, for all the cases hitherto have been fatal, with one exception, viz., that of Netter's,[1] which was cured by iodide of potassium.

The case I have recorded is also interesting in this respect, for the patient seemed to be improving so rapidly that I had great hopes of recovery, when death was brought about by the misadventure with chloroform.

TREATMENT.—The treatment is general and symptomatic, and in these respects need not be specially considered. When the actinomycosis is within

[1] Lemine, *Union Médicale*, 1893, p. 508.

reach, *i.e.*, on the surface, the abscesses may be opened and scraped, and in some instances cure has been effected, but recurrence in the neighbourhood is the rule.

Surgical treatment of the pleura or lung is, so far, unsuccessful, and is likely to remain so.

There is, however, one drug upon which great value is rightly laid, viz., iodide of potassium. This must be given in large doses. Upon actinomycosis of the skin there can be no doubt as to its effect, for it is obvious to the eye. One case of pleural actinomycosis is recorded,[1] in which the cure was complete; a second in which great improvement occurred; and in my own case, there seemed every reason to hope for cure, when death was unfortunately brought about by misadventure. What is well worthy of note in this latter case is, that the actinomycosis, which was found during life in the pus, was very difficult to discover in the diseased tissues after death; and these facts, taken in connection with the great improvement observed clinically, support the opinion that the iodide of potassium was directly destructive to the fungus.

A similar and very striking case is recorded by Dr. Galloway, in which, in a case of actinomycosis of the vermiform appendix, where the skin had been greatly involved, and many abscesses had formed, complete cure took place under the use of iodide of potassium. When the patient died subsequently, of some other affection, the *post-mortem* examination showed complete cicatrisation in all the diseased parts, and the most careful examination, by the microscope, failed to discover any trace of the fungus.

History.—It is to Böllinger in 1876 that the credit is due of assigning actinomycosis as a common cause of disease in cattle, though isolated cases had been described previously by other writers, *e.g.*, by Robin of 1871, and by Hann in Munich in 1870. Even as far back as 1845 appearances were described and figured which are now obviously those of actinomycosis, by Langenbeck of Kiel in a case of vertebral caries, and in 1848 by Lebert in a case of a man of 50 years of age, supposed to be suffering from cancer of the lung.

In man the disease was first described by Israel in 1878, but Ponfick in 1882 was the first to show the identity of the disease in man and cattle. Since then records of cases have been numerous. The first case in England was described in the liver by Acland and Sharkey, and soon after by Shattock; while Dr. H. Taylor's case at the Brompton Hospital, subsequently described by him and others at the Medical-Chirurgical Society, is the first instance in England in which the Ray-Fungus was discovered in the pus obtained from a patient during life, and this was a case of pulmonary actinomycosis.

Recent observation has shown that actinomyces is not the only pathogenic form of streptothrix met with in the lungs.

The following is the case which was under my own observation, and to which reference has been made above.[2]

Actinomycosis of the Pleura in a Child of Six.—Richard W., aged 6, was admitted into St. Bartholomew's Hospital under my care, as a case of empyema.

The whole left side was dull; the vocal vibrations, vocal resonance and breath sounds were absent over the lower part; and in the fifth intercostal space on the left side was a fluctuating swelling.

The cardiac dulness extended an inch to the right of the sternum; the respirations were 40, the pulse 160, and the temperature 102°.

The child was pale, feeble and ill, with considerable shortness of breath and slight cyanosis.

There seemed to be no doubt about the diagnosis of empyema, and an exploratory puncture obtained pus.

The history given by the mother was as follows:—

The child had been in its usual health until eight or nine weeks ago, and then complained of pain in the left side, and was put to bed. It remained in bed for six weeks, lying chiefly, as the mother said, upon the left side. The breath had been getting shorter, and the child

[1] Netter, *l.c.*

[2] *Path. Soc. Trans.*, vol. xlviii., 1897.

had lost flesh; there had been no shivering, and the swelling in the side had only been there a few days. On further inquiry, it appeared that twelve months ago the patient was laid up with bronchitis, and had some pain in the left side then, but had apparently completely recovered from that attack.

A few days after admission an incision was made into the pleura through the fluctuating swelling, and 6 ounces of curdy pus removed. The patient was very faint after the operation, and had to have brandy and ether. A few drachms of pus after this were discharged daily. Four days after the operation 6 ounces of pus escaped suddenly, and it appeared that in all probability another small collection of pus had burst through the opening.

The pus removed during the operation was examined by Dr. Kanthack, and appearances found which suggested the diagnosis of actinomycosis.

This was confirmed by subsequent examination.

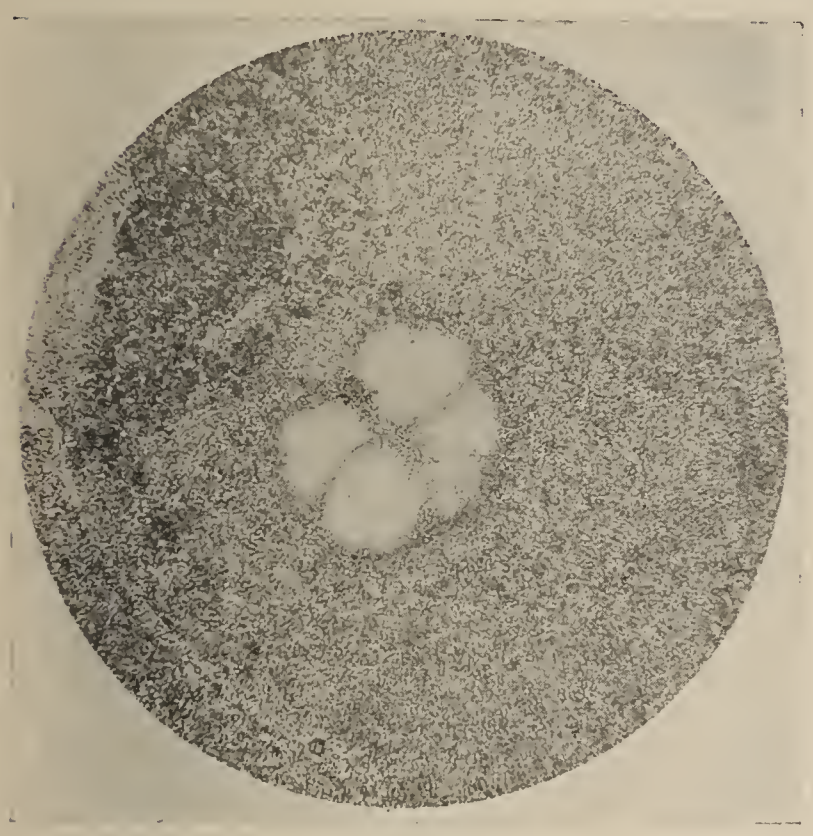


Fig. 170.

Actinomycosis, from the pus (*Kanthack*).

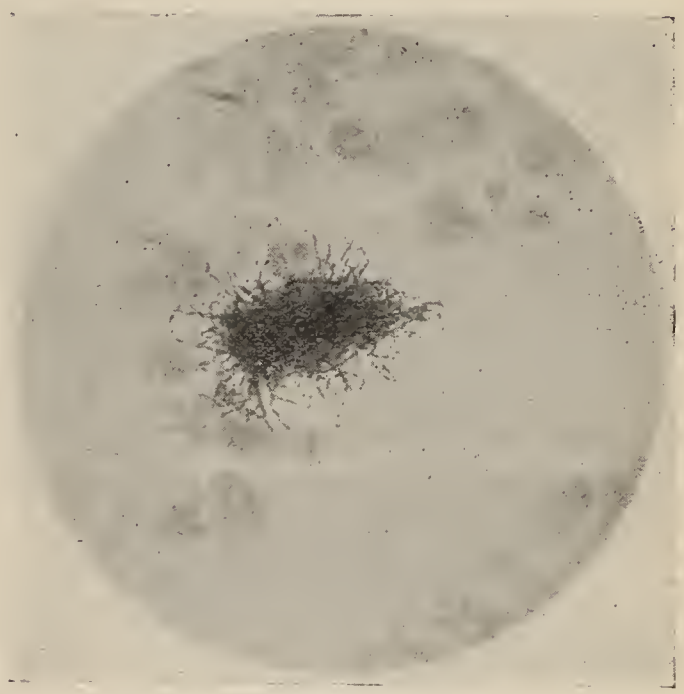


Fig. 171.

The same under a higher power (*Kanthack*).

The child did not seem materially relieved by the operation. The temperature, which had been of a hectic character, remained unchanged, and reached 103° or thereabouts every evening.

On 28th September, *i.e.*, sixteen days from the original operation, a new fluctuating swelling was discovered towards the front in the fifth intercostal space. This also was opened and a little pus discharged, but it did not seem to communicate with the pleura.

On 18th October, *i.e.*, three weeks later still, another abscess appeared nearer to the sternum. This also was opened. On all the occasions on which operations or incisions were performed the patient had chloroform, and took it well.

There were now three openings in the chest walls, one of which communicated with the pleura, all discharging the same kind of pus, though not in large amount, and showing no signs of healing.

The skin was a good deal undermined, and was syringed out daily. This gave the child a good deal of pain, and the patient was now put into a weak iodine bath daily, and was allowed to wash the chest and the skin wounds out by means of the respiratory movements. This gave great relief, and the patient seemed to be much improved.

The side now showed signs of considerable retraction; the hectic temperature, however, continued as before.

On 23rd November the patient was placed upon doses of iodide of potassium, which were rapidly increased, so that on 8th December the patient was taking 24 grains daily, and on 18th December 30 grains daily.

From the time that the patient was placed upon this drug improvement was marked, and although the temperature did not fall materially, the general condition of the patient improved greatly.

It lost the cachectic, anæmic look that it had and began to gain flesh, while the discharge from the wounds in the side became materially less.

By the beginning of January the improvement was so marked that I began to think the patient would probably recover.

On 4th January some chloroform was given for the purpose of extracting a tooth, and while under its influence the patient suddenly died ; why, it was hard to say, for it had previously taken chloroform three times for the other operations without any ill effects.

This was a great disappointment in every way, for the recovery had been so marked since the iodide of potassium had been administered, that I had almost made up my mind that the child would recover.

The following is the account of the *post-mortem* examination.

The body was thin. There were three wounds to the left of the sternum, two of them connected with superficial abscesses and the third leading into the pleura. The left side was considerably retracted. On removing the lungs, the right lung was found normal. Some enlarged glands were found at the bifurcation of the trachea, but these did not contain actinomyces.

The left lung was completely collapsed, and encased in a brawny, whitish material which occupied the whole pleural cavity.

In this numerous but small abscess cavities were found, from which yellowish pus was obtained. There were no naked-eye appearances of actinomyces visible in this tissue or in the lung. The brawny material extended into the posterior mediastinum, and was closely adherent to the aorta. It also extended between the ribs to the subcutaneous tissue, which was greatly thickened and indurated. The ribs were nowhere affected.

There were no secondary growths in any organs, nor any amyloid disease.

The chest walls were transformed into a dense cartilage-like tissue in which the ribs were firmly imbedded. It measured three-quarters of an inch in thickness. Microscopical examination of the most likely parts of this tissue showed nothing except firm fibrous tissue. No trace of actinomyces could be found. Some of the thickened brawny material from the pleural cavity, after prolonged search, revealed one or two characteristic fungus masses. It was remarkable how difficult it was to find the fungus in these places, in spite of the easy discovery of it in the pus during life.

The lung was carefully dissected to see if any trace of primary mischief could be found in it, but it appeared simply collapsed.

On deep dissection some small abscesses were found near the root of the lung, and in the pus obtained from one of these, typical actinomyces were found, from which the photograph was taken.[1]

Pseudo-Tuberculosis Hominis Streptotricha.

Under this name, Flexner[2] describes the case of a man who presented the constitutional and local signs of phthisis ; the autopsy, however, revealed lesions resembling those of tubercle, but containing no tubercle bacilli but a *streptothrix*.

The left lung was almost completely consolidated and gray in colour ; many parts were breaking down. Where the consolidation was not so dense, discrete tubercle-like nodules were seen. The lesions in the right lung were more focal and the nodules widely disseminated. Similar nodules covered the surface of the peritoneum.

Careful microscopical examination showed the entire absence of tubercle bacilli, and in their place clumps or convoluted masses, composed of a branching mycelium-like organism resembling actinomyces.

Streptothrix Israeli.

Until recently the actinomyces was thought to be the only pathogenic form of streptothrix met with in man. Recent observations show that there are others. The most important is that described by Wolff and Israel,[3] and since named Streptothrix Israeli. Morphological-, culture-, and inoculation-tests yield different results in the two cases, and justify their being regarded as distinct species.

An important paper has recently appeared, entitled, "Necrotic Broncho-pneumonia with Streptothrix," by Drs. Norris and Larkin,[4] describing two cases of the kind.

[1] *Cf.* figs. 132, 133.
[2] *Johns Hopkins Hosp. Bulletin*, 1897, No. 75.
[3] *Virch. Archiv*, 1891.
[4] *Jour. of Exper. Med.*, vol. v. p. 2.

1. M., 45 years of age, in previously good health, had suffered, for seven weeks only before his death, with cough, fœtid expectoration, and slight dyspnœa.

The patient having high fever (103–5°), and presenting the signs of consolidation of both lower lobes, the diagnosis was made of gangrene of the lung, following pneumonia.

Post-mortem the whole right lung was found completely solidified, and the left lung contained several small patches of consolidation.

2. M., 23 years of age, had been troubled with cough and mucopurulent expectoration for many years, but had remained in his normal health till six months before his death, when his

Fig. 172.

Section of lung, showing necrotic and suppurating bronchitis with streptothrix clumps within the necrotic material in a bronchus. Fibrin and cells are in the surrounding alveoli. (From Drs. Norris and Larkin's paper, *Jour. of Exper. Med.*, v. pt. 2. Pl. xvi.)

symptoms got worse ; his breath became short, and he began to lose flesh and strength. One month before death all the symptoms became still further aggravated, and he suffered from fever, with headache, repeated shiverings, and profuse sweatings. The physical signs were those of general bronchitis, with consolidation of the right lower lobe of the lung.

Post-mortem the right lower lobe and the left upper lobe were completely solidified, and numerous small patches of consolidation were scattered throughout both lungs.

In both cases the general changes in the lungs were much the same.

The mucous membrane of the trachea and bronchi was œdematous, intensely congested, and bestrewn here and there with whitish masses resembling "actinomyces granules." Bronchiectatic dilatations were numerous throughout both lungs.

The consolidation was firm, and resembled that of an organising pneumonia. The connective tissue septa were œdematous and grayish in colour.

The odour of the lung was fœtid, and myriads of small yellowish-white foci were scattered throughout both lungs.

The lesions consisted of an acute exudative and necrotic inflammation of the bronchi and surrounding alveoli, with inflammation and thickening of the interstitial tissue.

The examination of the whitish masses proved them to be formed of masses of filaments resembling actinomyces, though differing in many morphological respects from it.

An elaborate study was made of the fungus by cultivation and inoculation. The differences observed proved that the fungus was not actinomyces, but a different one, probably the Streptothrix Israeli, while the intense necrotic inflammation of the bronchi distinguished the pathological lesions produced by it in the lung, from those met with in the ordinary form of pulmonary actinomycosis.

For the full details the original paper should be consulted, where the literature of the subject is also given.

79. ASPERGILLOSIS.

Aspergillosis of the lungs is almost always non-pathogenic, and the result of the accidental injection of some pre-existing lesion. Most of these lesions are of a destructive nature—*e.g.*, gangrene, tubercular excavation, malignant growths, bronchiectasis, hæmorrhagic infarct—but it has been met with also in chronic bronchitis.

The form almost invariably met is the aspergillus fumigatus. The aspergillus niger (Fürbringer) and aspergillus glaucus (Osler) have been also described, but it is open to question if these forms are pathogenic (Sticker).

Fig. 173.

Aspergillus fumigatus, showing mycelium and fructification. (Wheaton, *Pathol. Tr.*, xli. p. 36.)

The figures show the essential structure of the fungus.

The form it presents in the body depends upon whether it grows on a free surface or within the tissues. On a free surface, the mycelium invades the tissues, and the fructification takes place from the surface. Within the tissues, the masses are composed of closely felted and compact mycelium only.

The fungus is derived chiefly from the fodder of cattle, in which it abounds. Many cases are described among hair-combers in Paris, and pigeon breeders. The latter feed the birds by taking the pap into the mouth and forcing it down the birds' throats.

The reactions excited by the fungus in the tissues are of the inflammatory kind, and vary according to the degree of intensity of the inflammation. Thus there may be produced more or less extensive consolidation of a pneumonic or broncho-pneumonic character, in parts of which necrosis or breaking down may occur. More often the inflammatory changes are of a less acute or even of a distinctly chronic character. They are then associated with the development of fibrous tissue; that is to say, an attempt at cure is made by cicatrisation, and if the attempt be successful, nothing may ultimately be left but a mass of fibrous tissue.

Fig. 174.

Small nodule of aspergillus, arising in the wall of an alveolus, and surrounded by macrophages and tissue-debris. (From *Jour. of Pathol.*, 1893. *Zeiss*, obj. $\frac{1}{4}$.)

The general lesions are thus much the same as those met with in actinomycosis.

In the inflammatory form, the areas of consolidation may measure half an inch to two inches in diameter. They often present, on section, a "honeycomb" appearance. These "honeycomb" spaces are sometimes small cavities, due to necrosis, but more often are merely greatly distended alveoli, the walls of which are densely infiltrated with the mycelium, while the lumen contains the fructification, which may extend into it in the form of rosettes.

SYMPTOMS.—The symptoms present nothing characteristic, but are simply those due to the reaction set up in the lungs. Where this is of the nature of an acute inflammation, the case may present itself clinically as one of pneumonia or broncho-pneumonia; more often the inflammation being subacute, the symptoms resemble closely those of phthisis, while in the most chronic cases there may be no symptoms at all.

The diagnosis in any case can only be made by the discovery of the organism either in the sputum during life, or in the organs after death. The diagnosis may be confirmed by cultivation and by subsequent inoculation into animals, for the details of which Renon's treatise may be consulted.

DURATION.—The duration is uncertain and variable. In acute cases it may be short and run the course of acute phthisis. In chronic cases the affection may last from three to eight years. In most of the secondary cases the lesions are accidental and independent of the disease they complicate, and the date of infection is quite uncertain.

PROGNOSIS.—As regards prognosis, all that need be said is that, in the secondary or accidental cases, its presence does not modify in any way the prognosis of the existing disease. The primary cases, however, appear to run a more rapid course, and to be of altogether graver significance, but fortunately they are very much rarer.

TREATMENT.—On general principles iodide of potassium and arsenic may be given, but of the actual results upon the disease we have even less evidence than in the case of actinomycosis.

The following cases are interesting illustrations of the subject.

In Boyce's case the aspergillus, though pathogenic in the sense of having caused lesions, had probably no effect upon the health, and was discovered accidentally in a patient who died of heart disease.

In the apex of one lung a few small irregular cavities were seen, in which were scattered small white resistent bodies, supposed at first to be calcified nodules, but which microscopical examination proved to be mycotic. The affected portions of the lung were hepatised. In the consolidation were small white points, and in other parts necrotic foci, which formed the small cavities. The walls of the cavities were formed of the remains of lung tissue, densely infiltrated with mycelium, and from the surface rose pigmented conidia spores, growing into the cavity. The neighbouring bronchi and small passages communicating with these cavities presented brownish patches similarly composed.

The white tubercle-like points in the hepatised tissue consisted of closely felted mycelium, some rounded, others reniform in shape.

In the two following cases the aspergillus seems to have been the actual cause of death. (*For references, see literature below.*)

Dr. Wheaton's case occurred in a girl 2½ years old, who had been ailing for two months with cough and loss of flesh. Consolidation was found at the right base in front, and at the right apex behind. A week later signs of breaking down showed themselves in the base. Eleven days after admission the temperature rose high, to 105° and 106°, and death soon followed.

On the autopsy, the lower three-quarters of the right lung were found consolidated, the consolidation being made up of patches of broncho-pneumonia, containing numbers of bright orange-coloured bodies, about the size of a mustard seed, which could be picked out with a knife. In these patches of consolidation numerous small cavities existed, and there was a larger cavity behind, the size of a Tangerine orange. The walls of the large cavity were black in colour, and covered with small white granules. In the main bronchi were several white gelatinous patches, and in the left lung a few broncho-pneumonic patches with orange-coloured granules.

The white patches in the bronchi were formed of mycelium, with rosette-like clusters of spores, and similar masses of mycelium and rosettes were found wherever the lung was breaking down.

Drawings are given showing the naked-eye appearance of the lung, and the microscopic characters of the fungus.

Arkle and Hind's case was that of a healthy man, aged 22, who became rather suddenly ill with shortness of breath on exertion, and some cyanosis. The physical signs were indefinite. He gradually got worse, and three months later was found to have some dulness at the left base behind. He suffered from attacks of pain, probably pleuritic, from time to time, and was slightly feverish. His chief trouble was dyspnœa, which came on in paroxysms, sometimes of considerable severity, in one of which he died, four months from the commencement of his illness.

Post-mortem the lungs were in a condition of well-marked emphysema. The base of the left lung was fleshy and partly consolidated, the pleura over it being thickened and inflamed. It presented the appearance of a sponge, containing numerous minute cavities, and between them, in the consolidated parts, small specks of a whitish colour. These proved to be masses of the mycelium of aspergillus fumigatus.

The following cases illustrate the close resemblance the affection sometimes presents to phthisis.

Potain records the case of a man who presented the signs of phthisis. No tubercle bacilli were found in the sputum, but aspergillus fumigatus in abundance. Four years later the case was examined by Renon, with the same result, and again, three years later, by Grancher, when tubercle bacilli were found as well. One year later still, *i.e.*, eight years from the commencement of the affection, when examined again by Renon, neither tubercle bacilli nor aspergillus were present.

A man of 25 years of age had had a cough since the age of 17, and presented signs of phthisis. Out of 21 examinations made of the sputum, tubercle bacilli were found in 2 and aspergillus in 18. A year later the patient died; the lungs showed cavities and much fibrosis, but neither tubercle bacilli nor aspergillus (Renon).

In the cases of a man, aged 37, and his wife, aged 40, the signs of chronic phthisis were present. The sputum was frequently examined, and showed the aspergillus repeatedly, but never tubercle bacilli (Renon).

History.—Attention had not been directed forcibly to these affections until Virchow described 5 cases in 1856. Up to that date a few isolated cases only had been recorded, one of the earliest of which was that reported by Gairdner in 1853. The next contribution of importance was that of Fürbringer in 1876, who brought the literature of the subject up to date. The most recent and complete treatise is that of Renon, published in 1897, which deals with the experimental as well as the pathological side.

Literature.—Virchow, *Virchow's Arch.*, ix., 1856. Fürbringer, *do.*, lxvi. 330, 1876. Weichselbaum, *Wein med. Woch.*, 1878, p. 1289. Baumgarten, *Lehrb. d. path. Mycologie*, 1886–90. Grawitz, *Virch. Arch.*, lxxvi. Freyhan, *Berl. klin. Woch.*, 1891, Dec. 14. Wheaton, *Trans., Path. Soc.*, xli. p. 34. Arkle and Hinds, *do.*, xlvii. p. 8. Boyce, *Jour. of Pathol. and Bacteriol.*, 1892, p. 165. Gilchrist and Stokes, *Bull. Johns Hopkins*, 1896. Flexner, *do.* Gilchrist and Rexford, *do.*, June 1897. Renon, *Aspergillosis*, Paris, 1897. Sticker, *Nothnagel Spec. Path. u. Ther.*, xiv. Saxer, *Pneumonomycosis Aspergillosa*, Jena, 1900.

Other Pathogenic Fungi of the Lung.

Mucor Corymbifer.—Fürbringer [1] describes a case of cancer of the stomach in a man of 66, with secondary growths. In the lung were two patches of consolidation the size of a walnut, with commencing gangrene, but without fœtor, in which mucor corymbifer was found.

Another similar case occurred in a man of 31.

In a man of 52,[2] who had long been subject to chronic gastritis, acute peritonitis developed, and death resulted with the symptoms of general pyæmia. *Post-mortem* numerous abscesses were found in the lungs and in the brain, as well as extensive ulceration of the intestines, and general suppurative peritonitis. All these lesions contained mucor corymbifer in abundance.

Oidium Albicans.—Grawitz [3] describes two cases, both in diabetics. The lungs presented numerous broncho-pneumonic patches and some gangrenous and fœtid cavities. The oidium was found in all. It also existed in quantity in the pharynx and œsophagus. Microscopically the mycelium could be seen growing through the walls of the small bronchi into the vesicles.

80. COCCIDIOSIS—PROTOZOAN (or COCCIDIOIDAL) INFECTION OF THE LUNG.

A case of Protozoan or Coccidioidal Pseudo-Tuberculosis is recorded by Reckford and Gilchrist.[4]

A man, 40 years of age, had suffered from a chronic slowly-spreading sore on the back of the neck for eight years. Similar sores developed on the face, especially the eyebrows and eyelids, and on one leg. About a year before his death the cervical lymphatics became enlarged. A few weeks before death the onset of cough, fever, and rapid emaciation, with signs of lung disease, suggested the diagnosis of pulmonary tuberculosis. On the autopsy, besides the skin lesions, nodules resembling tubercles were found widely disseminated through the lungs, over the peritoneal surface of the liver and diaphragm, in the spleen and suprarenal bodies, in the vesiculæ seminales, and in many of the internal lymphatic glands.

The lesions in the lungs closely resembled those of tubercle. Some of them were caseated, and in many small cavities had formed, containing débris and pus.

[1] *Loc. cit.*
[2] Paltauf, *Virch. A.*, 1885, cii.
[3] *Virch. Arch.*, 1877 and 1880.
[4] *Johns Hopkins Hospital Reports*, 1896, i. 209.

Microscopical section showed these nodules to be identical with tubercle in all respects save the presence of tubercle bacilli. There were numerous giant cells, and lymphoid cells with caseation and necrosis, but the place of tubercle bacilli was taken by the protozoa. These were abundant in all the necrotic cavities, whether in the lung or other parts of the body.

The original disease in the skin was protozoan, and the visceral lesions the result of the general dissemination through the body.

The paper contains many interesting details, and may well be consulted.

81. BILHARZIA HÆMATOBIA IN THE LUNG.

Fig. 175.—Bilharzia in the lung, from a preparation in the Museum of St. Barthol. Hosp. The oat-shaped body in the centre is one of the ova.

The specimen from which the sections were taken was presented to the Museum of St. Bartholomew's Hospital by Dr. Mackie (No. 2393*c*). It was obtained from the body of a country arab who was admitted into the hospital at Alexandria in a wretchedly emaciated condition, suffering greatly from his bladder, and passing bloody urine, which swarmed with the bilharzia ova. He died shortly after of chronic uræmia. The autopsy showed the bladder, ureters and pelvis of the kidneys to be infested with the ova, as were also the lungs, liver, and spleen. The whole urinary organs are preserved in the museum, and portions of the latter organs, as well as several specimens of the parent worms taken from the portal vein.

There is no full clinical account given of the case, but presumably what drew attention to the lungs was hæmoptysis.

The specimen preserved shows no marked naked-eye changes, but on microscopical section the tissues are seen to be infested with the ova. They lie in the interstitial tissue, in which they have produced no change but slight connective tissue proliferation, the surrounding alveoli being normal.

82. HYDATIDS OF THE LUNG AND PLEURA.

Hydatids of the lung and pleura are rare in this country, but they are more frequent where hydatid disease is prevalent, as in Australia.

The lung and pleura are not favourite seats of hydatids.

The relative frequency with which the different parts of the body are affected with hydatids is shown by the following figures.

Combined Statistics of Davaine, Cobbold, Finsen, and Neisser.[1]

Liver,	953	51 per cent.
Intestinal canal,	163	8·8 ,,
Lung or pleura,	153	8·3 ,,
Kidneys, bladder, and genital organs,	186	10·0 ,,
Brain and spinal cord,	127	6·9 ,,
Bone,	61	3·4 ,,
Heart and blood vessels,	61	3·4 ,,
Other organs,	158	8·2 ,,
	1862	

The lung or pleura is shown to be affected in about 8 per cent. of all cases, and to stand towards the liver in respect of frequency in the proportion of about 1 to 6.

Thomas's[2] figures give a proportion somewhat less than this, of about 1 to 4.

Lung,	16·56
Liver,	65·76

Osler,[3] in 85 American cases, found the lung or pleura affected in 6, or 1 in 14.

The pleura is much less frequently the seat of disease than the lung, and according to the statistics of Thomas, the lung is affected eleven times to the pleura once.

Hearn's[4] earlier figures show a smaller difference.

Lungs,	57
Pleura,	16
Mediastinum,	2

The cysts in the lung and pleura, as in other organs, are usually single; occasionally there are two or three, either in one lung or in both, and in a few remarkable cases small cysts in considerable numbers have been found.

To these reference will be made again later.

The cysts may be simple, *i.e.*, barren, or proliferating. Proliferation is usually endogenous, that is to say, the cysts develop one within the other. It has been stated that exogenous proliferation is sometimes met with, but I have not been able to find any conclusive case of the kind recorded.

In the lung the cysts may develop in any part of it, but are usually found in the lower lobe, and most frequently on the right side.

Thus, of 53 cases, Hearn states that 27 occurred in the right lung, 13 in the left, and 13 in both.

In the pleura primary hydatids are very rare. In most instances the hydatids are secondary, that is to say, the cysts have developed in organs near, and have ruptured into the pleura.

In other cases, what appears to be a hydatid of the pleura is really a hydatid cyst of the lung or of the chest walls,[5] which has developed towards the pleura, and has pushed the pleura in front of it without penetrating into the pleural cavity.

[1] *Encycl. d. Sc. Méd.*, art. "Echinococcus."
[2] *Hydatid Disease*, Adelaide, 1884; also *Australian Med. Jour.*, 1889.
[3] *Practice of Medicine*, 1895.
[4] *Kystes Hydatiques du Poumon et de la Pleura*, Paris, 1875.
[5] Lesser, *Deutsch med. Woch.*, 1881, No. 1, "Peripleuritic Hydatid." Otto, *St. Petersb. Med. Woch.*, 1884.

The structure of the hydatid itself is the same as in other parts of the body; the only peculiarity in the lung is that the fibrous sac surrounding the cyst is unusually thin. This is remarkable, for as the cyst develops, it of course condenses the lung tissue surrounding it; but as the cyst wall remains so thin, it follows that considerable atrophy of the condensed lung tissue must take place.

When a hydatid develops into the pleural cavity, it acts like a pleural effusion and pushes the lung aside. Some of these pleural hydatids may reach a very large size.

Thus Leroux[1] records a case in which the cyst contained 6 litres (210 oz.) of fluid, besides numerous secondary cysts.

In the lung the cysts do not, as a rule, reach such large dimensions, although they may sometimes occupy the whole of a lobe, and there are one or two rare instances in which the cyst has occupied the whole lung. Usually, long before the cyst has reached so large a size, it ruptures; but rupture may take place even when the cyst is still quite small.

RESULTS.—A hydatid, as long as it is growing, is not a particularly irritating body.

In the lung it causes atrophy of the surrounding tissues rather than any inflammatory irritation, so that the fibrous cyst-wall which results is peculiarly thin; neither does it cause definite symptoms, unless the cyst is of great size, or so placed as to cause pressure. It is usually not until the hydatid has perished, and the cyst become inflamed, that serious symptoms begin to arise.

In the pleura, also, or in its immediate neighbourhood, the cysts may grow without any reaction at all being produced in the pleura, so that they may reach a large size. When a serous effusion develops, the hydatid cyst may be found floating in it, as in a remarkable case recorded by Trousseau.

In another case, recorded by Reboul,[2] there were two fluctuating tumours on the outside of the thorax, with signs of effusion in the pleura. The diagnosis was made of tubercular pleurisy, with cold abscesses in the chest walls. The case proved to be one of hydatids, and on incision, besides the hydatids in the chest walls, two others were found in the pleura, one simple, the other proliferating, and containing two litres of fluid, with a large number of secondary cysts. Operation was performed and the patient cured.

As a rule, if pleuritic effusion occur in association with hydatids, it is because they have ruptured into the pleura.

The effusion is, then, nearly always purulent and the prognosis very grave.

If a cyst in the lung die, in most cases, especially if the cyst be a proliferating one, secondary changes occur in it which lead to suppuration. If suppuration do not occur, the fluid may be absorbed, the sac shrink, and the membrane become folded; lime salts may then be deposited in it, and in the course of time a mass of a cheesy appearance be left, in which the folds of membrane may be distinctly traced, or the characteristic hooklets found many years after. A completely calcified cyst in the lung, such as is met with in the liver, is, however, not recorded.

Rupture.—Sooner or later, if the cyst continue to grow, it spontaneously ruptures; but rupture may be determined sometimes by mechanical causes, as, for example, by a fall or a blow on the chest, and in these instances the rupture may be the first evidence of the existence of a hydatid at all.

[1] *Cf.* Hearn, *l.c.*, Case vii. Chvostek, *Oesterr. Ztsch. f. pract. Heilk.*, 1867, No. 37.
[2] Hearn, Case xxi., *Gaz. d. Hôpitaux*, 1893, No. 131.

Theoretically, rupture might occur into any part near. In the lung it is most likely to take place into a bronchus, and this happens in at least half the cases.

When the cyst ruptures, it might be supposed that the fluid and contents would be suddenly discharged in large quantities into the air-tubes, and thus dangerous suffocative symptoms be produced. This does occur sometimes, but, as a rule, the rupture is not so sudden; it takes place rather by a process of gradual oozing; in other words, the communication with the bronchus is not a large one, at any rate at first. If the cyst be barren, and contain a large amount of fluid, even a small perforation may lead to its complete evacuation, and that in a very short space of time, and it is in these circumstances that sudden symptoms of suffocation are likely to arise. With a proliferating cyst the aperture is soon filled by one of the smaller hydatids, which gradually squeezes itself through; another takes its place, and then another, and so on, so that the evacuation of a cyst of this kind may take a considerable time, and the patients continue to spit up small hydatids or portions of membrane for many months.

What more frequently happens is, that before the cyst is emptied, suppuration occurs; and this may be of a fœtid or putrefactive character.

As soon as the cyst is discharged, whether after suppuration or not, a cavity is left; this may continue to suppurate for some time, but in the end it may become quiescent. When all traces of the hydatid have been removed, it would be impossible to say, except from the history, that the cavity had owed its origin to a hydatid, as in an interesting case recorded by Habershon.[1]

Hæmoptysis is not an uncommon occurrence with hydatids of the lung. It very often precedes or accompanies the expectoration of the membrane, but at these times it is not usually large in amount. Hæmoptysis may, however, be profuse, and is sometimes fatal. It is then brought about in the same way as profuse or fatal hæmoptysis in a chronic tubercular cavity, either by ulceration into a pulmonary vessel or by the rupture of a pulmonary aneurysm.

A case of the latter kind is recorded by Habershon.[2]

A lad, aged 17, had been spitting blood from time to time for $2\frac{1}{2}$ years, sometimes in considerable quantity. Some hydatid membrane had been found in the expectoration, and the diagnosis made of a hydatid situated at the base of the left lung.

The hæmorrhage recurred frequently, and the patient was admitted into the hospital, where he died soon after of exhaustion consequent on the repeated loss of blood.

Post-mortem a small cavity, the size of an orange, was found in the base of the left lung in the position suspected, lined with caseous material, but with no trace of hydatid membrane, so that without the history of the membrane having been discovered in the expectoration during life, no diagnosis could have been made. The hæmorrhage was due to the rupture of a small aneurysm on a branch of the pulmonary vein.

After the bronchi, the next commonest place for rupture is the pleura. Rupture into the pleura has been followed by sudden death from shock; under any circumstances it is attended with severe symptoms at the time and with serious consequences later; for violent inflammation is at once set up and an empyema develops, which often becomes of a septic character.

If the cyst, when it bursts into the pleura, has already also communicated with the lung, air, as well as the fluid and contents of the cyst, may reach the pleura, and thus hydro-pneumothorax be produced. In either case, the only prospect of cure is by incision.

A case of this kind is recorded by Lebert,[3] but the diagnosis of the cause, viz., rupture of a hydatid cyst, was only made on *post-mortem* examination.

[1] *Guy's Hosp. Rep.*, xviii. 375.
[2] *Guy's Hosp. Rep.*, 1872.
[3] *Berl. klin. Woch.*, 1872, Nos. 3 and 4.

If a hydatid of the lung or pleura rupture externally, it is only after suppuration. It points and bursts like an empyema, from which the diagnosis can only be made by the appearance of cysts, or portions of membrane in the discharge.

After suppuration, with hydatids, as with empyema, the pus may track in peculiar directions, and rupture take place in strange places. Thus there are cases recorded in which the pus has perforated the diaphragm, and the discharge taken place at the umbilicus,[1] into the stomach,[2] or into the intestines.[3]

Alibert[4] records a case in which rupture took place into the pericardium without previous suppuration. In this case the hydatid was seated in the right pleural cavity. The pericardium was filled with clear hydatid fluid, the patient dying before inflammatory reaction had time to take place.

There are some rare instances in which hydatids are found in large numbers irregularly disseminated through both lungs.

In some of these cases the hydatids occupy the interior of the vessels, and have evidently been carried by the blood-stream from some primary source either within the heart itself or near it.

The first case of this kind is recorded by Andral.[5]

A man, aged 55, was admitted into the hospital as suffering from morbus cordis; he had severe dyspnœa, and finally died of gradual failure of the heart.

At the autopsy, both lungs were found to contain a large number of hydatids of varying sizes, from that of a nut to that of a pea.

On careful dissection, these were found to lie, not in the lung tissue, but within branches of the pulmonary veins. Some of the cysts were perfect, others were ruptured, and the membrane lay folded up within the vessel.

A hydatid cyst, as large as a big orange, was found in the liver, with cartilaginous walls, and containing several daughter cysts.

Another interesting case of a similar kind is recorded by Budd.[6]

A woman, aged 23, was admitted into King's College Hospital with palpitation and dyspnœa, which she had been suffering from for about two years. She had occasional attacks of hæmoptysis; the symptoms got worse and worse, and, finally, she died with the signs of a failing heart.

On the *post-mortem* a hydatid, the size of an orange, was found in the walls of the right ventricle; this was filled with secondary cysts. A small hydatid was found attached to the tricuspid valve, and another just above the valve, in the pulmonary artery. Besides these, the branches of the pulmonary artery in the left lung contained numerous hydatids, chiefly in the upper lobe, one or two in the middle lobe, and a single one in the lower lobe. They varied in size from an eighth to a quarter of an inch in diameter. There were none in the pulmonary vein or bronchi.

Ducastel[7] records a case of numerous hydatids in the lung, associated with hydatids in the wall of the heart.

The patient, a young man of 25 years of age, had been troubled for three years with hæmoptysis, and had brought up on many occasions portions of hydatid membrane.

He was very pale and emaciated, and had a good deal of rhonchus and crepitation over the whole of both lungs, with pleuritic friction here and there, but no absolute dulness. He finally died of exhaustion.

On *post-mortem* examination the pleural cavities were obliterated on both sides, and the lung was full of cysts, some of which were visible on the surface and many more found on section. They all proved to be hydatids scattered irregularly through the lung, more at the base than at the apices; the largest were found at the posterior part of the right lung, and were of the size of a hen's egg; some had discharged themselves through the bronchi. Round many of the cysts was a certain amount of induration in the pulmonary tissue, but not much. In the right ventricle a hydatid cyst existed, containing a dozen or more secondary cysts within it.

[1] Dupuytren, *Leç. orales*, iii. 379.
[2] Neisser, *Die Echinococcus-kranh.*, 1877.
[3] Laennec, *Tr. d'Ausc.*, ii. 201.
[4] *Cf.* Hearn, Case ix.
[5] *Clin. Med.*, ii. 412.
[6] *Trans. Path. Soc. Lond.*, x. 80.
[7] *Bull. Soc. Anat.*, 1870, p. 363.

There is no statement made in the record of this case as to whether the cyst was located in the blood vessels or not.

Peacock[1] records the case of a young sailor, aged 18, who was admitted into the hospital with a good deal of pain on both sides of the chest, and a swelling of the right hypochondrium, and subsequently of the abdomen. Towards the end of his illness he spat up a considerable quantity of blood-stained pus, and subsequently some hydatid membrane. He died of exhaustion.

At the autopsy, the two lungs were found to contain numerous hydatid cysts, for the most part of the size of a chestnut; the bronchi contained pus. There is no further description of the exact seat of the hydatids. In the left ventricle a large collapsed hydatid cyst was found; the liver was greatly enlarged, and contained a very large hydatid cyst, with numerous daughter cysts; there were also cysts in the spleen as well as in the psoas and iliacus muscles.

In Wunderlich's[2] case there were numerous hydatids in various abdominal organs; the largest, in the liver, had perforated the pericardium, and there was one hydatid found in a branch of the pulmonary artery.

The case was that of a young man, aged 22, whose chief symptoms were abdominal. He had severe tumours, to be felt in different parts of the abdomen, and finally died of exhaustion and dyspnœa.

In the abdomen numerous hydatids were found, viz., in the spleen, in the liver, and in the peritoneal cavity.

The cyst in the liver was of a very large size, and contained a considerable number of secondary cysts. The hydatids had suppurated and perforated the pericardium.

In the inferior lobe of the right lung, in a branch of the pulmonary artery of the third order, was found a hydatid cyst the size of a pigeon's egg.

These cases are all remarkable, and the dissemination is evidently due to the direct passage of hydatids into the blood vessels. In many instances the source of the hydatids is obvious, for cysts were found in the walls of the ventricle itself. In the case of the liver, presumably the communication was with a branch of the hepatic vein. In other cases the source could not be traced.

SYMPTOMS.—The onset is generally insidious; there may be for a long time no symptoms at all. Even when the cyst is a large one, all symptoms may be absent, if it be growing slowly and not in a position to produce pressure on the root of the lung or other important part.

It may be that the very first symptoms are those produced by rupture of the cyst, *e.g.*, dyspnœa, hæmoptysis, or the expectoration of hydatid fluid or membrane. These may not only be the first symptoms, but may even rapidly prove fatal.

Thus in a case recorded by Krause,[3] the patient was thought to be in good health until he suddenly expectorated hydatid fluid and membrane. Eight days later he died.

The usual symptoms are shortness of breath, cough, and pain.

Shortness of Breath.—This may be of rapid onset or of gradual development; it may vary a good deal from to time, and sometimes be paroxysmal.

If severe dyspnœa suddenly set in, it is probably associated with the rupture of the cyst, and due either to the presence of hydatid fluid or membrane in the air-tubes, to hæmoptysis, or possibly to pneumothorax.

Sudden dyspnœa, if not connected with rupture, is probably caused by some inflammatory complication in the lung on the one hand, or by effusion into the pleura on the other. The latter is the most likely, for the cyst, so long as it is living, seems to have very little irritating effect upon the surrounding lung-tissue.

The most severe paroxysms of dyspnœa arise when large portions of hydatid membrane find difficulty in passing through the larynx.

[1] *Trans. Path. Soc. Lond.*, xv., 1864.
[2] *Arch. f. Physiol. Heilk.*, 1858, N.S., ii. 283.
[3] *Virch. Jahrsber.*, 1880, ii. 182.

In many cases the development of shortness of breath is gradual, and is directly connected with the size of the cyst.

A very large cyst may be present without much dyspnœa if its growth has been gradual, while a small cyst may cause much dyspnœa if it press on important parts, such as the root of the lung.

If, however, the cyst be growing rapidly, dyspnœa will be an almost certain symptom.

Cough.—In the early stages there may be no cough at all. If there be a cough, it is usually dry at first, *i.e.*, without expectoration, and of an irritative character. If severe and paroxysmal, it probably indicates impending rupture of the cyst.

If rupture have already taken place, the cough is due either to the irritation produced by the presence of hydatid fluid or membrane in the air-tubes, or to the bronchitis which has followed.

When large pieces of membrane find their way into the air-tubes, the cough may occur in severe paroxysms, and be attended with a considerable amount of dyspnœa. If the membrane have any difficulty in passing through the larynx, the cough and dyspnœa will be extreme, and if it become impacted there, the paroxysm will in all probability be fatal, as in a case recently under observation at St. Bartholomew's Hospital.

Sputum.—In the early stages, before rupture, there will be no expectoration at all, unless the cyst irritate the neighbouring bronchial tubes and produce a mucopurulent expectoration, bronchitic in character.

If the cyst be near a large bronchus or the trachea, and threatening to perforate it, a considerable quantity of a saliva-like fluid, perhaps streaked with blood, may be expectorated, as happens also under similar circumstances with new-growth or aneurysm.

When inflammatory changes have taken place in the cyst, the sputum will have the corresponding character; thus, if suppuration occur, the sputum will be purulent and copious, and resemble the discharge from an empyema or chronic pulmonary cavity; if putrefactive decomposition occur, the sputum will be fœtid; and if there be any destructive process in the neighbouring lung, it may contain elastic fibres or portions of lung tissue.

The sputum is only pathognomonic when it contains hooklets or portions of hydatid membrane. Membrane is easy enough to find, and can only be confused with the casts of plastic bronchitis, but the structure of the membrane will quickly decide between them.

Hooklets are much more difficult to find, and have not, as a matter of fact, often determined the diagnosis.

Of course the sputum will change its character according to the changes which take place in the cyst; so that in any given case, all the various characters mentioned may be met with as the disease runs its course.

Hæmoptysis.—With hydatids of the lung, hæmoptysis may not occur for a long time, but it is not absent throughout, except in about a quarter of all cases; with hydatids of the pleura, it does not, of course, occur at all, unless they perforate the lung. It varies in amount greatly; in early cases there is often nothing more than slight streaks of blood; but rupture is generally attended with more or less bleeding, and this may be profuse or even fatal.

Thus in a case recorded by Kidd,[1] a woman, aged 42, had had slight hæmoptysis on and off for a period of four months; she then expectorated some hydatid membrane, and died of profuse hæmoptysis.

[1] *Trans. Path. Soc. Lond.*, xxxvi. 122.

In Habershon's case, previously referred to, the fatal hæmoptysis had a different explanation; it was not directly due to the cyst, but was the consequence of an aneurysm which had developed in the chronic cavity, left by a hydatid cyst after its expulsion.

The most remarkable thing about the hæmoptysis, with pulmonary hydatids, is its long continuance.

Thus in Kidd's case, just referred to, the hæmoptysis persisted uninterruptedly for a period of four months. In another case, recorded by Curnow,[1] the hæmoptysis lasted for the same period, and was often dangerously profuse, though in this case the patient ultimately died of exhaustion and not of hæmoptysis.

Pain.—Actual pain is not a common phenomenon in the disease, but if it occur, and be well marked, it is probably due to an affection of the pleura.

What the patients complain of generally, rather than pain, is a feeling of discomfort, of tension, of weight, or of oppression in the chest.

Temperature.—The temperature depends on the complications; it is no part of the original disease. It may be the result either of some inflammatory complication on the side of the lung or in the pleura; more commonly it depends upon suppuration in the cyst itself. It is like the usual suppurative fever, hectic in character, and often attended with considerable sweating.

PHYSICAL SIGNS.—A hydatid does not necessarily affect the general health at all; but if complications occur, either in the cyst itself, or in the lung or pleura, the ordinary signs of such complications will present themselves; if, for instance, suppuration takes place in the cyst, there will be the usual signs of fever, hectic and sweating, and the case may come to resemble one of phthisis or of empyema.

The **decubitus** varies, as would be expected, and is not in any way characteristic.

The **physical signs** in the chest vary according to the size of the cyst; if it be small and deep-seated, there may be no physical signs at all; if it be large, the first sign given will probably be an area of percussion dulness.

As hydatids assume a more or less spherical form, the area of dulness will take, to some extent, the same shape, and though closely resembling in this respect a localised empyema, it will differ, on the one hand, from the more horizontal lines obtained with ordinary pleuritic effusion, and on the other with the more indefinite lines of pulmonary consolidation.

In some cases the shape of the cyst itself is masked by changes which have taken place in the lung or pleura round it.

Over such an area of dulness the vocal vibrations, vocal resonance and breath sounds will be absent, or, at any rate, greatly diminished; the movements also over the same parts will be impaired or perhaps absent. If the tumour be of large size, there may possibly be bulging; but this is not often met with, except where the cyst has suppurated, and is inclined to point externally. If the cyst be near the heart or liver, it may cause displacement of these organs, and greatly increase the difficulties of diagnosis.

In the same way, if the tumour be so placed that it can press upon important structures, it may give evidence of such interference by the usual pressure signs, *e.g.*, dyspnœa, defective entry of air in certain parts, dysphagia, difference in the pulses, etc. Such symptoms are most likely to be associated with hydatids of the mediastinum; but as intrathoracic hydatids generally develop within the substance of the lung, or in the pleural cavity, pressure signs are but rarely met with.

[1] *Trans. Path. Soc. Lond.*, xxxiv. 24.

When complications are associated with the cyst, the physical signs, of course, will vary; thus, if inflammations take place round the cyst in the lung, we shall find the signs of consolidation; if the bronchial tubes become inflamed, the signs of bronchitis; if inflammation of the pleura occur, the sign of effusion of serum or pus, as the case may be.

Lastly, certain miscellaneous symptoms have been described as of occasional occurrence; but, being not constant, they are of no great importance. Thus *clubbing of the fingers* has been observed,[1] but this is probably dependent upon some chronic fibroid change in the lung or pleura; at any rate, it is very rare and accidental.

Urticaria has also been described, especially when the cysts have ruptured or paracentesis has been performed. There is nothing peculiar in this, for it is met with under similar circumstances in a variety of other affections.

DIAGNOSIS.—It is evident from what has been said that the diagnosis of a hydatid is often a matter of very great difficulty.

In the majority of cases that are recorded, the condition has not been diagnosed during life, but only discovered *post-mortem*.

Sometimes, by the peculiar shape of the area of dulness, and by a gradual exclusion of other affections to which the physical signs might be due, a probable diagnosis may be arrived at, which may then be confirmed by the result of an exploratory puncture.

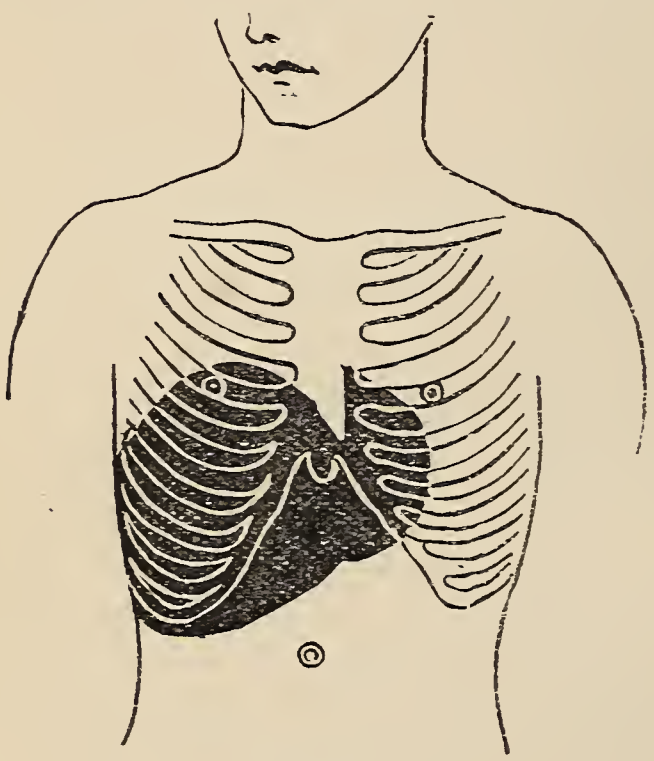


Fig. 176.

The accompanying diagram is from a case of hydatid of the lower part of the right lung. In this case, of course, the cyst, so far as the mere area of dulness was concerned, might have been in the liver, between the liver and the diaphragm, in the pleura, or in the lungs. In the chapter on Localised Empyema, a case is given which yielded a similar area of dulness.

If, in such a case, jaundice were present, or if, after rupture, the sputum contained bile, it would be fair to conclude that the liver was the prime seat of the cyst; but with hydatid of the liver, jaundice is usually absent, unless it has developed in such a position as to press upon the common duct, *i.e.*, in the portal fissure.

Even when jaundice is present, the contents of the cysts are not bile-stained, so long as the cyst is living.

Hydatid cysts of the kidney, and also of the spleen, have been known to develop towards the thorax and burst through the lung.

Usually the diagnosis is not made until rupture has taken place. Hydatid membrane is so characteristic that it cannot be mistaken, if it be expectorated, unless it be confused with plastic bronchitis; but very little care is necessary to prevent this confusion, for the laminated character of the hydatid membrane on section is utterly unlike that of a cast in plastic bronchitis, and the shreds of hydatid membrane are ragged and irregular, and not ramified or dendritic as are usually the casts of plastic bronchitis.

[1] Trousseau, *Clin. Med.*

In the absence of anything characteristic in the sputum, the diagnosis may be made either of phthisis or of empyema.

The resemblance to phthisis is of course very close; a large number of the cases have been diagnosed as phthisis during life, when the cyst has ruptured and suppurated, for there are in both the same progressive pulmonary symptoms, associated with hectic fever, purulent expectoration, and occasional hæmoptysis.

Careful examination of the sputum might decide the diagnosis if hooklets or pieces of membrane, on the one hand, or tubercle bacilli on the other were found, but if not, the diagnosis would remain undecided.

Where the pleura is the seat of disease, or where it has become secondarily involved, the signs will be those of pleuritic effusion. Hydatid fluid is, of course, easily distinguished from serous effusion, by the complete or almost complete absence of albumen, and by its low specific gravity, so that in the rare event of there being a hydatid only in the pleura and no effusion, the diagnosis could be made by exploratory puncture, but if a serous effusion were present too, there would be no means of determining its cause.

As already stated, the effusion is not always purulent, but may be serous, and in one remarkable instance a hydatid cyst was found floating free in a large serous effusion.[1]

From a localised empyema the diagnosis is difficult and often impossible. In many cases the diagnosis has only been made after incision, when hydatid cysts or portions of membrane have been discovered in the pus evacuated.

From new-growths of the lung and pleura it is obvious that there might sometimes be difficulties of diagnosis, but I do not know that they often arise.

DURATION.—The duration of hydatid disease of the lung it is quite impossible to determine accurately. If a hydatid become stationary or quiescent, it may last for years; but this is not nearly so likely to happen in either the lung or the pleura as it is in the liver, though it is not really frequent there. In some instances hydatid cysts have been found *post-mortem* when there had been nothing to suggest their existence during life.

In most cases the cyst continues to develop, and ultimately ruptures; the course and duration then largely depend upon the complications to which the rupture leads.

Rupture may cause sudden death by suffocation, by hæmoptysis, or by shock.

If the contents of the cyst were suddenly discharged into an air-tube, it is obvious that very serious symptoms would arise, and in all probability the patient would die at once of suffocation, but fortunately this does not often happen. The rupture is rather a gradual oozing, or if the cyst be a simple one, and contain a good deal of fluid, though the greater quantity is discharged at once, it does not result in any great distress, and the urgent symptoms quickly pass off. Many of the hydatid cysts, however, contain numerous secondary cysts, and one of these soon plugs the aperture, and is only gradually squeezed out of it; another then takes its place, and so on; the cysts thus pass into the air-tubes slowly one by one, and in this way may not produce anything more than paroxysms of coughing; but if a large cyst, or a large portion of hydatid membrane, get into the tubes, even if it pass rapidly through the larger tubes, it may cause considerable obstruction in the larynx, and even lead to a fatal suffocative attack. This, however, does not often appear to occur.

Death from hæmoptysis has already been referred to, but it is due, as a rule, to exhaustion rather than to suffocation.

[1] Trousseau, *Clin. Med.*, 1873, p. 845.

Where perforation takes place into the pleura, or into the pericardium, it is possible that sudden death may occur from shock, but for the reasons already given, it is less likely to happen with hydatids than with pneumothorax.

Usually the rupture of a hydatid, whether into the lung or pleura, leads to complications, which involve a long illness, and ultimately cause death from exhaustion.

Cases of short duration, like that of Krause,[1] in which death occurred eight days after the first symptom of illness, *i.e.*, from the date of rupture of the cyst, are very rare.

If gangrene occur in the cyst, septic symptoms arise, which, in a short time, may carry the patient off.

Spontaneous cure may occur either by the death of the hydatid or by its expectoration.

The death of the hydatid, with shrinking, shrivelling and ultimate calcification, is, as already stated, very rare in the lung, and unknown in the pleura.

Expectoration of the hydatid, consequent on its rupture into a bronchus, is, on the whole, a not unfavourable result, for it appears that, in the end, after a longer or shorter illness, about 70 per cent. of such cases recover.

Thus of 133 cases in which rupture took place into a bronchus,[2] 31 died and 80 recovered, the results of the remaining 22 being unknown. Compared with general statistics, this is a most favourable result. Taking all cases together, about 61 per cent. died, *i.e.*, of 208 cases, 113 died, 71 were relieved or cured, and of 24 the result was unknown.

TREATMENT.—The diagnosis is the chief difficulty; but, in discussing treatment, we will suppose the diagnosis to have been made satisfactorily, either on general principles or after preliminary puncture.

The administration of drugs internally is as useless in the case of hydatids of the chest as of other organs. A few cases have been recorded in which cure is supposed to have resulted from the inhalation of certain volatile drugs, such as ether or chloroform; but these probably rest upon incorrect observation, or are accidental coincidences.

The chances of spontaneous cure are practically nil, and rupture brings with it dangers of its own. The most favourable cases, as already stated, are those in which spontaneous rupture takes place into the bronchus, and the hydatid is expectorated.

Attempts to kill the hydatid by the injection of certain fluids, like tincture of iodine, or by electrolysis, usually result in failure, and introduce fresh dangers of their own.

The only treatment that offers a prospect of success for hydatids of the lung and pleura is surgical, viz., paracentesis or incision.

Paracentesis.—Simple puncture and the drawing off of a certain amount of fluid rarely succeeds in curing a hydatid in the lung or pleura in the way that it sometimes succeeds in the case of a simple hydatid of the liver. It can only succeed in those cases in which the hydatid is barren, *i.e.*, in which it contains no secondary cysts.

Simple puncture, so far as I have been able to ascertain, has not proved successful of itself, and is not altogether devoid of risk. In some cases, immediately following the puncture, rupture has taken place into a bronchus, and the patients have died of suffocation, either at the time or within a few hours of the puncture.

[1] *Wien med. Woch.*, 1880, No. 28. [2] Thomas, *Austr. Med. Jour.*, *l.c.*

Thus in the case of a child, 9 years of age, recorded by Bristowe,[1] a puncture was made through what was thought to be a pleural effusion; a few drachms of fluid only were obtained. Severe dyspnœa set in, and the child died of suffocation. On the *post-mortem*, a hydatid cyst was found in the left lower lobe, which had ruptured into the bronchus.

A similar case occurred at St. Bartholomew's Hospital, and is recorded by Wallis.[2]

A similar case is published by Mackenzie,[3] in which, after exploratory puncture, the patient died with sudden dyspnœa the next day.

In another case, recorded by Leclerc and Tillier,[4] the left pleura was punctured for what was thought to be an empyema; a few days later a second puncture was made, when the patient died suddenly with paralysis of the right arm, for which no cause could be found *post-mortem*.

Sometimes, after puncture, the cases have recovered, either by expectoration of the membrane or after subsequent incision.

The two following cases are good examples of this.

Revillod[5] records a case of a man, aged 19, who had had shortness of breath and cough for two months; the physical signs pointed to what was thought to be an effusion in the right pleura; puncture was made, and 1150 c.c. of hydatid fluid, with plenty of daughter cysts, were withdrawn. Paracentesis was stopped, because of cough and albuminous expectoration. Subsequently the fluid became purulent; the cavity was then incised, a large amount of membrane and pus evacuated. In three months the patient was cured; but after the incision a communication took place also with a bronchus, so that hydatid membrane was expectorated as well as discharged through the incision.

Incision.—There is no doubt, if the diagnosis can be correctly made, and the hydatid cyst accurately localised and safely reached, that free incision through the chest walls offers the most favourable prospect of cure.

Of 38 cases treated by free incision, recorded by Thomas,[6] 32 recovered, and in some of these, suppuration or even gangrene had previously occurred.

In most of these cases, of course, the diagnosis had been previously determined after the rupture into a bronchus and the expectoration of hydatid membrane. Rupture into a bronchus is no contra-indication, but rather an indication for incision, if the cyst can be properly localised.

Thus in a case recorded by Israel,[7] in a woman of 25, a hydatid was localised in the lower part of the right lung; it was punctured, but spontaneous rupture into a bronchus also took place at the time of paracentesis; 1500 c.c. of fluid and membrane were coughed up. Two days later resection was performed and free drainage of the cavity made through the right pleura. With the exception of a little broncho-pneumonia, which occurred after the operation, the patient made a good recovery.

Many other cases of a similar kind are recorded.

When the incision has been made and the cavity reached, every part of the membrane that can be obtained should be freely removed, and the opening made free, so that any portions of membrane left behind may find an easy exit; for if any of it remain, it is almost certain to putrefy or gangrene, and become the source of dangerous septic infection.

Ord[8] records a case in which, after operation upon a hydatid of the lower lobe of the lung, a fœtid empyema and purulent pericarditis developed, as the result, no doubt, of general pyæmic infection.

A very successful case has been recently recorded by Steiner.[9] It occurred in a country girl, who, a year and a half before pulmonary symptoms developed, had had the liver opened for a hydatid, which had been evacuated and cured; then physical signs developed on the left side of the thorax; puncture was performed, and hydatid fluid containing hooklets withdrawn. A

[1] *Clin. Soc. Trans.*, vol. xxiv.
[2] *St. Barth. Hosp. Rep.*, 1892, p. 239.
[3] *Clin. Soc. Trans.*, 1892, p. 215.
[4] *Lyon. Méd.*, 1889, No. 36.
[5] *Rev. d. l. Suisse Romande*, Août 1890.
[6] *Loc. cit.*
[7] *Deutsch med. Woch.*, 1886, No. 19.
[8] *Clin. Soc. Trans.*, xxiv. 125.
[9] *Centralbl. f. Chir.* 1898, No. 1.

free incision was made at the left base behind, and parts of the seventh and eighth ribs resected; a large hydatid was removed, and rapid recovery took place. Six weeks later, dulness appeared at the right base, and, on puncture, hydatid fluid was also obtained; dyspnœa immediately followed, and the patient coughed up hydatid fluid in large quantity. This was followed by a certain amount of broncho-pneumonia, but in the end all symptoms disappeared, and the patient became perfectly well. Some contraction took place on the right side, where the puncture only was made, but on the left, where the free incision was made, no deformity resulted.

The fear of operating on these cases, through a pleura which has not been obliterated by adhesions, is not justified by experience; for, if the drainage be free, it frequently happens that no secondary infection of the pleura of any consequence occurs, and the operation is not followed by the disastrous results which had been theoretically anticipated; on the contrary, recovery frequently takes an uninterrupted course.

A good many cases of hydatid cysts of the liver have been treated in this way by incision through the thorax; thus Thomas has brought together the records of 17 such cases; 12 of these recovered completely; 5 only died, 1 of these deaths was due to a pneumonia which had begun before the operation, another to hæmorrhage from a gastric ulcer, while not one of the remaining 3 died of any pleural affection.

This is remarkable, when we bear in mind the disastrous results which follow the spontaneous rupture of hydatids of the liver into the pleura, even when operation has been performed early; for in 8 cases of this kind, to which Thomas refers, the result was fatal in 6.

83. NEW-GROWTHS OF THE LUNGS AND PLEURA.

New-growths in the trachea and main bronchi have been already dealt with, and need not be again referred to.

What remain to be considered here are new-growths of the lung and pleura. These are either innocent or malignant.

The cases upon which this account is based consist of 155. Of these 56 are taken from the published records in the Pathological Society's *Transactions*. The rest are derived, some from other published records, some from the *post-mortem* and clinical records of St. Bartholomew's Hospital, many of them not yet published, and some from private sources.

The innocent tumours are very rare, and are of more pathological interest than clinical importance. The malignant are not so uncommon; they are, for the most part, secondary to malignant disease in some other part of the body, involving the lung and pleura either by direct extension from parts near, or by metastasis.

NON-MALIGNANT TUMOURS.

The innocent tumours of the lung are Fibroma and Osteoma, common to both pleura and lung; Enchondroma, peculiar to the lung; and Lipoma, peculiar to the pleura.

Fibroma.—Fibroma **of the lung** is a name we hear but rarely nowadays. Formerly it was given to small, discrete, fibrous masses, sometimes in considerable numbers, and widely disseminated through one or both lungs, or to large masses limited to a part of one lung. The origin of the masses is often not to be definitely traced, but in many instances they are the result of past inflammatory or destructive disease; thus, the fibromata which are peribronchial

are usually the result of tubercle, as are most of the more diffuse fibrous changes in the lung, and some fibrous nodules may be consolidated pulmonary aneurysms. Where the fibrous tissue is more diffuse, it may be the result of some of the pneumo-konioses, or, where it involves a whole lobe, of a past pneumonia. A good many instances of this kind have been already referred to under the heading of Fibrosis or Fibrotic Affections of the Lung. Eliminating cases of this class, it is difficult to find a conclusive instance of pure fibroma or fibrous tumour of the lung of an innocent nature.

So also with **the pleura.** Fibrous thickening is almost always the consequence of past inflammation.

The post-inflammatory thickening may be very considerable, so that the pleura may measure an inch or more in thickness in parts, and a condition may be produced in the pleura which may be fairly compared with keloid in the skin. This has been sometimes described as Callous Pleurisy (*Pleuritis callosa*).

I have come across only one case of fibrous disease of the pleura which seems to deserve the name of Fibroma. This is the case recorded by Charlwood Turner.[1] It consisted of "a pedunculated body of loculated form, somewhat resembling a piece of coral, attached to the base of the lung."

Lipoma. — Lipoma is the name given to small, roundish masses of fat tissue, usually beneath the pleura or between the lobes of the lung. They are very rare.

Osteoma.—Both in the lung and pleura new-growths of true bone may be met with, but they are pathological curiosities.

In the lung they occur either as irregular spicules, or as nodules or rounded masses; in the pleura as thin plates.

In either case they have to be distinguished from calcareous deposits in pathological tissues, which are common enough.

The only instance I know of true osteoma of the lung is described by Virchow,[2] in a woman of 70, in whom an irregular spicular mass was found in the upper lobe of one lung. Calcareous deposits, not bone, of irregular shape, are sometimes found in the walls of chronic cavities, or in the fibrous tissues of the lung round about; others may be small bronchi or small blood vessels which have become calcified[3]; the more nodular masses are usually calcified lymphatic glands or tubercular nodules. One case of calcified enchondroma is described by Förster.[4]

Enchondroma.—Enchondroma, though very rare, is the most interesting of all the primary growths in the lung. These tumours originate from the cartilage of the bronchus, and form small rounded masses, usually not larger than a pea or a cherry. They produce no symptoms, and are only discovered accidentally *post-mortem*.

Among the cases collected, there are only 6 of enchondroma, and this is sufficient evidence of the rarity of the affection. Of these six, 2 reached a considerable size, and as they also involved the glands at the root of the lung and on the mediastinum, they were probably not simple enchondromata, but rather of the nature of chondro-sarcoma.

These 6 cases all occurred in males, and at various ages, viz., 28, 39, 44, 64, 66.

[1] *Path. Soc. Trans.*, xxxiv. 19.
[2] *Geschwülste*, iii. 100.
[3] Luschka, *Virch. Arch.*, x. 550.
[4] Luschka, *Virch. Arch.*, xiii. 105.

MALIGNANT TUMOURS.

The malignant tumours are Cancer and Sarcoma.

In both cases alike they are generally secondary, and involve the lung or pleura either by direct extension from the parts near or by metastasis, the former being the most common with Sarcoma and the latter with Cancer.

Where the growth spreads by direct extension, it invades all tissues alike.

The metastatic growths, no doubt, develop in most cases from infective particles carried by the blood, and are embolic in origin. Then they spread from a centre, radially and concentrically, so that the resulting tumours are more or less round.

When the infection spreads by the lymphatics, the form of the tumour is different; thus, in the pleura the new-growth forms flattened masses of, perhaps, no great thickness, but, possibly, of wide extent. At the root of the lung it extends in a radiating fashion for some distance into the lung, following the divisions of the bronchial tubes.

Dissemination may sometimes take place by the air-tubes from particles derived from a primary growth in the throat or large air passage, *e.g.*, with epithelioma of the larynx or trachea, or even of the tongue.

In many cases these different modes of extension are variously combined.

In the case of the pleura, the new-growth may extend, as it were, by simple contact; thus, where the parietal pleura has become involved, the visceral pleura opposite may be found affected without any adhesion between the two, and with apparently healthy serous surfaces.

New-growths may sometimes spread, too, through a tissue without obviously involving it; for instance, the diaphragm may be covered with growths on both the pleural and peritoneal surfaces, and yet its own tissue be apparently unaffected.

New-growths, whether cancer or sarcoma, are more common in the lung than in the pleura, as would be expected, for most of them are metastatic, and metastasis is rare in the pleura. Yet there are a few instances in which the pleura has been the only seat of metastatic infection, and the lung has entirely escaped.

SARCOMA.

Sarcoma is a much rarer form of tumour, both in the lung and pleura, than cancer. Thus, out of the cases collected, there were only 15 cases of sarcoma; yet this by no means represents the relative preponderance of cancer.

Sarcoma spreads rather by direct extension than by metastasis, and, except in the mediastinum or neck, the parts about the chest are not favourite seats of primary sarcoma. Even when the primary growth is near the chest, it often leads to death before the pleura or lungs have had time to become much involved.

Putting aside the cases in which the lung or the pleura has become affected by direct extension from the parts about, sarcoma of the lung or pleura is a rare disease, and it is, with few exceptions, secondary.

Thus, of the 15 cases, the primary growth existed in bone in 5 cases, in the liver in 2, in the kidney in 1, in the testicle in 1, in the eye in 1, so that only 5 cases remain in which the growth was of primary origin.

Sarcoma of the pleura alone is extremely rare. There is one remarkable instance of it recorded by Coupland.[1]

[1] *Pathol. Soc. Trans.*, xxvii. 23.

In this case the whole left side of the chest was occupied by a tumour, the lung being compressed and pushed aside, but not invaded. The primary growth was a spindle-celled sarcoma of the sciatic nerve, and the secondary growths had developed in the pleura and mediastinum.

Recent cases are recorded by Blumenau [1] in a man of 23, and by Finlay and Bradley [2] in a man of 67. The last authors refer to 6 other published cases.

In the lung the secondary tumours are, as a rule, of the same nature as the primary growth, *e.g.*, osteo-sarcoma, chondro-sarcoma, or melanotic sarcoma, yet they may be of a simple nature, *i.e.*, they need not reproduce the special characters of the original growth. Thus, in a case of osteo-sarcoma, the secondary growth may be quite soft, and contain no bone or calcified deposit.

The lung appears to be the favourite seat of secondary osteo-sarcoma, and is not infrequently the only seat of secondary growth in the body.

The most striking fact about these cases of secondary sarcoma of the lung is the long interval which may elapse from the time that the original tumour was operated on before pulmonary symptoms develop. Thus there was an interval of two and a half years in one case after removal of the humerus, and four years in another case after removal of a testicle. In other cases, however, the interval may be short, a few months only, or a year at most.

Though sarcoma is, in general, the commoner form of malignant disease in the young, this rule does not seem to hold in the case of the lung and pleura.

Thus, among the ten secondary cases, there were only two instances in young persons, at the age of 17 and 18 respectively; the rest were all of adult or advanced life. Even the primary cases were distributed over all periods of life, and were not confined to the young, the ages being 18, 26, 42, 45, 46.

The secondary cases do not call for much comment.

The following case, which was under my own care, is a fairly characteristic, though in some respects a noteworthy, instance.

Charles T., aged 18, a carman, was admitted into the Chest Hospital on account of dyspnœa. He was found to have a large pleuritic effusion on the right side, which proved to be sanguineous. Nine and a half months previously a tumour had developed in the left tibia, which had been removed by operation, and was found to be a myelo-sarcoma. The patient recovered completely after the operation, and remained perfectly well until a fortnight before I saw him. He had been attacked at that time with pain in the left side and shortness of breath, and, a few days later, had some palpitation of the heart.

He lay in a semi-recumbent position, was pale and somewhat cyanosed, and suffered with a good deal of dyspnœa, especially on movement.

The physical signs were very remarkable, as is shown in the diagram. The whole front of the body, from an inch below the umbilicus to the second rib on each side, was absolutely dull, and all breath- and voice-sounds absent except at the apices, where the vocal vibrations and resonance were increased, and the breath sounds somewhat exaggerated.

There was slight bulging of the right side. The respiratory movements were costal in type, fairly free and equal on the two sides, but the abdomen was motionless and diaphragmatic respiration entirely absent. Tracheal breathing was distinct all along the sternum, even to the xiphoid cartilage, though fainter here than elsewhere. On the left side there was an area of resonance in the mid-axilla which was thought to be stomach. The only portion of the left lung which seemed to be performing its function was the lower part of the left lung behind.

The pulses were equal, but feeble; the temperature was slightly raised to about 100°, but there were no other signs worthy of notice.

Two days later the patient's side was tapped, and 48 ounces of sanguineous fluid, almost as bright as blood, removed. More could not be taken away, because the patient complained of pain and became faint.

The patient gradually sank, and died two days later, just ten months after the tumour had been removed from the tibia.

[1] *D. med. Woch.*, 1896, No. 14.

[2] *Practitioner*, Aug. 1897.

On *post-mortem* examination, the liver was found depressed and rotated, so that the notch lay under the left costal arch, and the gall-bladder two inches to the left of the umbilicus.

The right side of the diaphragm was convex towards the abdomen.

The right pleura contained several pints of blood-stained fluid. The right lung was completely collapsed and airless; the pulmonary pleura was roughened with numerous fleshy vegetations, but the parietal pleura was quite smooth, except in one place corresponding with the seventh rib, two inches from the spine, where there was a lobular, spongy mass, as large as a Tangerine orange, growing from it, but perfectly disconnected from the parts beneath.

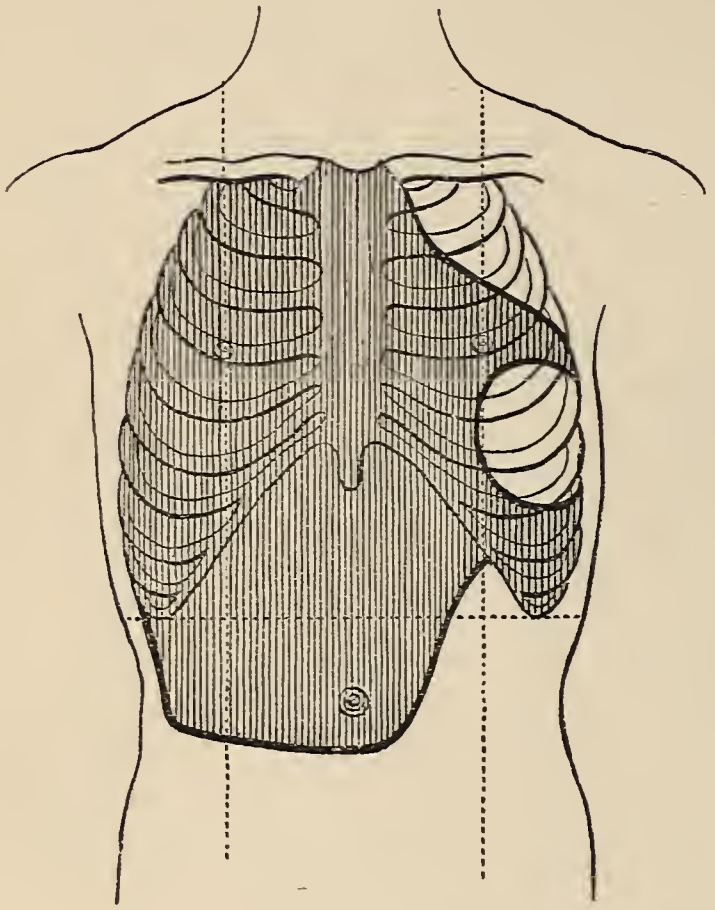


Fig. 177.

Diagram of the case of secondary osteo-sarcoma of the lung, showing the remarkable extent of dulness in the front of the chest. (See further description in text.)

The lung was irregular in shape owing to the several tumours in it, one of which occupied the whole mediastinum.

Between the lower lobe and diaphragm, but attached to the lung, was a mass the size of a cricket ball, covered with dark, laminated blood clot.

The pericardium contained a little serous fluid, but no new-growth.

In the left lung a large mass pushed the pericardium in front of it, and occupied the whole of the adjacent portion of the upper lobe, forming an irregular, oval tumour 6 inches by 4½ inches. In the rest of the lobe were four or five independent nodules situated near the surface, and, like the larger mass, of white colour. The lower lobe contained one medium-sized growth and four or five small ones.

The bronchial glands were not involved.

The two layers of the pleura were universally adherent on the left side, but could be easily separated.

The stomach was greatly displaced and twisted, so that the whole greater curvature was in contact with the left side of the thorax, and clearly accounted for the area of resonance previously referred to.

There were no secondary deposits elsewhere. The stump of the femur was carefully examined and the bone divided, but there was no thickening or evidence of tumour in the stump or neighbouring glands.

The tumours in the lung appeared so soft and spongy that some hesitation was felt in making sections of them, lest the preparations should be spoilt for preservation. Some surprise was, therefore, felt on cutting into them to find that they were so hard that the knife could hardly divide them, and even the smallest of them offered considerable resistance. To the naked eye they presented the typical appearances of osteo-sarcoma, as they proved to be on microscopical examination. The tumours, however, contained no myeloid cells, though in the primary tumour of the tibia these cells existed in large numbers.

The case is remarkable, first, in respect of the extraordinary physical signs; secondly, in respect of the large size which the tumours had reached, and their number; thirdly, in the fact that the patient had but a small portion of one lung left to breathe with; and, lastly, in the fact that the patient had been free from symptoms, and thought himself to be well until one month before his death, when the symptoms set in suddenly, and rapidly led to suffocation.

Another somewhat similar case occurred at St. Bartholomew's,[1] in the year 1895, in a girl of the age of 17, in whom large tumours developed in the lungs, secondary to chondro-sarcoma of the femur, which had been amputated some months previously.

The tumours in the lung were like those of the femur, viz., chondro-sarcoma.

The right lung was almost completely invaded by the tumour, except just a little round its root. In the left lung there was a growth, the size of a Tangerine orange; in the apex, and in the lower lobe another mass, hard and calcareous, which had invaded the pleura and the diaphragm. There were a few small nodules also in other parts of this lung.

[1] *P.-m. Reg.*, xxii. 210.

A case, recorded by Prentiss,[1] deserves mention on account of the long period that elapsed between the removal of the original growth, a sarcoma of the testicle, and the development of the symptoms in the chest and death of the patient.

After the removal of the testicle the patient remained in good health for four years, when he began to suffer pain in the right side of the chest, had a cough, and some blood-stained sputum. The side was explored with a needle, and the diagnosis of sarcoma made by the examination of portions of tissue removed by that means.

The patient lived for twelve months longer, so that five years elapsed between the original operation and the death of the patient at the age of 51.

The whole right lung was found to be converted into tumour, and a cavity existed at the base containing 2 ounces of pus. There were no other secondary growths elsewhere.

The cases of **primary sarcoma of the lung** deserve some further reference on account of their rarity. I have only been able to find records of 5 cases.

1. A case in a man of 46, recorded by Wilkes,[2] in which the tumour is described as a fibro-cellular growth in the base of the left lung.

2. A case in a man of 18, recorded by Davies,[3] whose illness began, nine months before his death, with cough and pleurisy. He suffered from night sweats, loss of flesh, pain in his left side, and occasional hæmoptysis. Subsequently some glands grew above the clavicle, and the patient died fifteen months from the commencement of his illness.

Post-mortem the whole left lung was found involved in the new-growth, which extended through the diaphragm, and projected into the abdomen. A mass was found above Poupart's ligament of some size. A few secondary growths were found in the liver, but none elsewhere.

The tumour was described as a round-celled lympho-sarcoma, and it was believed to have originated in the lung.

3. A case, in a male of 42, described by Spillman and Hanshalter,[4] in which the symptoms resembled cardiac disease.

A large sarcoma was found in the upper part of the left lung, measuring 7 inches by $4\frac{1}{4}$ inches.

4. A similar case in a man of 26, recorded by Kronig,[5] in which there was a primary sarcoma in the right lung, and this was diagnosed early by exploration with the needle.

5. A recent case at St. Bartholomew's Hospital, which was under the care of Dr. Hensley.

CANCER.

Cancer of the lung and pleura is much commoner than sarcoma.

The general frequency of cancer in these parts is shown by Reinhardt's[6] statistics. Thus, out of 8716 autopsies, cancer was the cause of death in 545, and among these the lung was affected 74 times, *i.e.*, of the cases of cancer the lung was affected in 13·6 per cent.

In most cases the affection of the lung is secondary and of quite subordinate importance. It is a part of the general dissemination of cancer, and growths are often found in the lung without having given any evidence of their presence during life.

Primary cases of cancer of the lung and pleura, according to Reinhardt's statistics, bear to the secondary the proportion of about 6·8 per cent., but this is probably too high. At any rate, cancer of the lung and pleura appears to be the cause of death in not more than 1 per thousand of the total mortality.

It is usually stated that the relation of the sexes in primary growths is equal, but that, in secondary growths, cancer of the lung and pleura predominates among women, the reason given being that cancer of the breast and of the pelvic organs is more common in the female. Figures, however, do not seem to bear this statement out.

The forms of cancer met with vary to some extent according as the growth is of primary or secondary origin, and according as it is the lung or pleura which

[1] *Med. News*, July 1893.
[2] *Path. Soc. Trans.*, ix. 31.
[3] *Ibid.*, xl. 46.
[4] *Gaz. hebd.*, 1891, No. 48.
[5] *Berl. klin. Woch.*, 1887, No. 51.
[6] *Arch. f. Heilkunde*, 1878, xix. 369.

is chiefly affected. Colloid and melanotic cancer never affect the pleura at all except by direct extension; *e.g.*, in the lung are, without exception, secondary to similar growths elsewhere; in the abdominal cavity especially in the case of colloid, and in the eye or skin in the case of melanotic cancer.

Epithelioma, again, is, as a rule, secondary to growths of this nature in the larynx or trachea, or in the tongue. It may also develop in the lung by metastasis, the primary growth being in a distant part of the body.

Thus Godlee[1] records the case of a man of 78 who was the subject of epithelioma of the bladder. A secondary growth of some size, the only one in the body, was found *post-mortem* in the lower part of the left lung—a typical epithelioma with cell nests. Arnott[2] also describes an instance in a woman of 50, secondary to epithelioma of the clitoris. In this case there was very extensive involvement of the pleura as well.

The form of cancer that is most frequent in the lung is the medullary, *i.e.*, the growth in the lung is soft and contains little fibrous tissue, even when the primary growth has been scirrhus.

There is one exception, however, to this rule, and that is when the growth is primary at the root of the lung. In primary root cancer, as also in primary cancer of the pleura, there is generally a large amount of fibrous tissue.

Although the lung and pleura are usually affected together, still they may be affected independently of one another, and that when the growth is secondary as well as when it is primary. As growths of the lung and pleura present many pathological as well as clinical peculiarities, it will be convenient to discuss them separately.

Again, in cancer of the lung the clinical problems differ according as the new-growth affects the body of the lung or its root.

The subject, therefore, divides itself conveniently into three parts—cancer of the pleura, cancer of the body of the lung, and cancer of the root of the lung.

Cancer of the body of the lung may be primary, but is, in the great majority of cases, secondary.

Secondary Cancer of the Lung.

Cancer may involve the lung secondarily either (1) by direct extension from the parts near, or (2) by infection (*a*) through the blood vessels, (*b*) through the lymphatics, and (*c*) lastly, through the air-tubes.

In all cases its seat in the lung is interstitial, and the alveoli, as the tumour grows, are at first compressed by it before the walls become actually involved.

1. By direct extension from the parts near, *i.e.*, from the neck, mediastinum, chest walls, or abdomen.

In these cases the growth in the lung is likely to be localised and unilateral, whereas in the cases of infection the secondary growths are usually irregularly disseminated more or less through both lungs.

Where there is an external or palpable tumour, this group of cases presents little interest, for the symptoms are subordinate to those of the primary growth.

Where, however, the primary growth is deep-seated, as, for example, in connection with the vertebræ, lymphatic glands, or mediastinal tissues, the pulmonary or pleural symptoms may be among the earliest to attract attention. The diagnosis then becomes very difficult; but the difficulties are the same as with primary cancer of the lung, from which the diagnosis is often impossible during life.

[1] *Path. Soc. Trans.*, xxxii. 30. [2] *Ibid.*, xxi 231.

In most cases of direct extension, the different layers of the tissues are involved one after the other, until the lung is reached; but in some instances the affection seems to spread through one tissue without involving it much, if at all.

For example, the diaphragm may be found covered with new-growth on both its serous surfaces, the peritoneal and the pleural, and yet the tissue itself may have escaped. No doubt the infection in these cases travels by the lymphatics.

2. By infection:

(*a*) *Through the blood vessels.*—As in the case of embolic infarcts, the masses are multiple and irregularly scattered through the whole of both lungs. Each spreads from a centre and grows concentrically, so that its shape is more or less rounded.

Usually the number of secondary tumours is not very great, and, judging by their different sizes, they must be of different ages; that is to say, the infection must have taken place at different times.

Occasionally the masses are very numerous, and minute or even miliary in size, so as to resemble in general appearance miliary tuberculosis, and on the analogy of tuberculosis these cases have been described as *Carcinosis of the Lung.* The condition is a very rare one, but it may develop in the course of a primary tumour of the lung, just as in the course of a primary tumour in any other part of the body.

A case of this kind occurred at St. Bartholomew's Hospital.[1] A girl of 16 suffered with cancer of the bones of the head and face. When she died, the lungs were found studded with minute nodules of new-growth, and there were many of small size also disseminated in the various organs of the body.

A similar case is recorded by Risdon Bennett,[2] in a girl of 15. She suffered for three months with what seemed to be general bronchitis, with loss of strength and flesh. She died after five months' illness. Many tumours were found in the liver, lumbar glands, spleen, and kidney, with miliary dissemination in the lungs.

Similar cases are also recorded by Fagge[3] and Bristowe.[4] Fagge's case occurred in a man of 50. The case had been regarded during life as one of bronchitis, with more or less pneumonia.

Post-mortem serous fluid was found in both pleural cavities, and also some new-growth in the pleura. Both lungs were studded with nodules, the largest being the size of hemp-seeds.

There were also several nodules of small size in the pericardium, in the substance of the left ventricle, and also in the liver and spleen. The new-growths were soft, and thought to be primary in the lung.

Bristowe's case[5] occurred in a woman of 36, whose lungs were studded with small nodules of cancer. There were three or four small growths also in the liver, but none elsewhere.

Of the 4 recorded cases, the first 2 were obviously secondary, and the 2 last were regarded by the authors as primary.

(*b*) *Through the lymphatics.*—In the case of a primary abdominal cancer, when the lymphatic glands about the spinal column are affected, the new-growths may extend upwards in the course of the lymph stream, pass between the pillars of the diaphragm, and so involve the lymphatics of the mediastinum. In many of these cases the extension takes place from the affected glands direct to the lung, or it may extend to the lung along the root. In the same way the lymphatics of the mediastinum or root of the lung may be infected by a cancer of the neck.

Again, with cases of cancer of the peritoneum or of the liver, the infection may spread from the abdominal surface through the diaphragm to the pleura and lung, and sometimes without any obvious change in the tissues of the diaphragm itself. The infection in this case, no doubt, takes place through the lymphatics.

[1] *Post-mortem Registers*, vol. xx. p. 115.
[2] *Intrathoracic Tumours*, p. 13.
[3] *Path. Soc. Trans.*, vol. xvii. p. 29.
[4] *Intrathoracic Tumours*, p. 13.
[5] *Path. Soc. Trans.*, vol. xi. p. 35.

In an interesting case at St. Bartholomew's Hospital, in a woman of 36, who died with general cancer of the peritoneum and liver, secondary to cancer of the rectum, the diaphragm was covered on both sides with masses of growth, as were also the corresponding parts of the lower lobes of both lungs, and that without adhesion between the pleural surfaces. Yet the muscular tissue of the diaphragm itself appeared to be quite unaffected.

The same may occur in the walls of the thorax, and thus, after cancer of the breast, the parietal pleura may be found widely involved without direct connection being traced to the pleura through the chest walls.

The most interesting mode of infection through the lymphatics is that in which the new-growth spreads across the pleural cavity without any adhesions or direct connection between the two surfaces, as in the case just cited. The most striking instances of this occur in connection with cancer of the parietal pleura. Then there may be found on the part of the lung opposite a network of lymphatics infiltrated with cancer, and yet both the pleural surfaces may be perfectly smooth and without adhesions through which the infection could have directly passed. No doubt the infection in such a case is carried by the lymphatics across the pleural cavity, and is picked up by the stomata on the surface opposite.

Lastly, it is possible for the infection to spread up along the lymphatics to the thoracic duct, and thus be introduced into the veins. This may account for some of the instances of wide dissemination, especially in the lungs.

(*c*) *Through the air-tubes.*—This is an important mode of secondary dissemination in the lungs, especially in the case of some of the rarer forms of tumour. Thus, instances of true epithelioma of the lung are described in connection with new-growths in the larynx or trachea, and occasionally also with new-growths of the pharynx or tongue; and again, in cases where a new-growth in the lung itself, or near it, has communicated with a bronchus, it is often through the air-tubes that the dissemination takes place.

Godlee[1] records a good instance of epithelioma of the lung following a primary growth in the tongue. The tumour in the lung presented perfectly characteristic appearances, and contained typical cell nests.

A similar case is recorded by Hutchinson,[2] secondary to the tongue, and another by Broadbent,[3] secondary to a growth in the larynx.

Primary Cancer of the Lung.

Primary cancer of the lung, whether of the body or of the root, is usually unilateral. It often remains a single mass, though by secondary infection from the primary growth others may develop in the lung, or even general dissemination occur. It is with cancer of the body of the lung that the largest masses are found; for then the growths may invade the whole of the lung, and the mass weigh several pounds.

E.g., 90 oz. (Suckling), 6 lbs. (Graves), and 9 lbs. (Lobstein).[4]

With cancer of the root, however, the mass is generally small, and may be easily overlooked; but it is in this case that the more marked secondary changes occur in the rest of the lung, owing to the interference of the growth, small as it is, with the bronchi, blood vessels, and lymphatics at the root.

Sex.—Of 61 cases of primary cancer of the lung, 47 occurred in men and 14 in women, a proportion of about 3 to 1.

[1] *Path. Soc. Trans.*, vol. xxxii. 27, 30.
[2] *Ibid.*, vol. xii. 46.
[3] *Ibid.*, vol. xii. 44.
[4] *Lancet*, Dec. 13, 1884.

Hasse[1] gives 17 males to 5 females; Reinhardt, 16 males to 11 females; together giving 33 males to 16 females, or a proportion of 2 to 1; and Köhler's statistics give a proportion of 5 to 3. Though the actual figures differ somewhat, all show the same relative preponderance of primary cancer of the lung in males.

Age.—The ages are given in the following table:—

Table showing the Ages of Cases of Primary Cancer of Lung.

—20		—25		—30		—35		—40		—45		—50		—55		—60		—65		—70 and above.	
M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
2	2	2	...	3	1	1	...	6	1	5	1	7	1	4	2	7	2	5	2	5	2
4		2		4		1		7		6		8		6		9		7		7	
(Reinhardt), 4						2				8				2				8			

These tables show that cancer of the lung, like cancer of other organs, is more frequent after 40 than before, in the proportion of about 3 to 1.

Only 4 cases occurred under the age of 20, and of these all, except 1, at the age of about 17 or 18. The youngest case on record is that reported by M'Aldowie,[2] in a child of $5\frac{1}{2}$ years.

Seat.—The seat is shown in the following table:—

	Right Side.	Left Side.
Upper,	9	4
Middle,	5	1
Lower,	4	8
Whole lung,	2	3
	20	16

Of 60 cases, it affected the root in 17 and the body of the lung in 43.

It is more common on the right than on the left side, in the proportion of 5 to 4.

The combined statistics given by Wilson Fox[3] show the right lung to have been affected in 49 cases and the left in 32.

Of the cases of cancer of the body of the lung, the upper part was affected in 13 cases, the middle in 6, the lower in 12, and the whole of the lung in 5.

Two cases are recorded by Wolf in which the tumour developed in a chronic tubercular cavity. Friedländer had also described a similar case. Cancer and tubercle were held by Rokitansky to be incompatible. This is not so, for Wolf, in 31 cases of cancer of the lung, found tubercular lesions also in 13.

Two cases are recorded of **primary carcinosis of the lung,** the growths being in one case minute and miliary, and in the other of somewhat larger size or nodular.

[1] *Loc. cit.* [2] *Lancet*, 1876, ii. 570. [3] *Loc. cit.*

The nature of the growth.—In primary cancer of the body of the lung, the growth is nearly always of the medullary type; but at the root of the lung scirrhus, *i.e.*, it contains much fibrous tissue.

I only know of one case recorded of primary epithelioma of the lung.[1] This was a growth of large size, seated in the right, middle, and lower lobes, showing typical epitheliomatous structures with cell nests.

Primary Cancer of the Root of the Lung.

This is of the fibrous form, and is usually described as cancer; recently some of the cases have been called sarcoma—large-celled or alveolar sarcoma. This is probably merely a difference of terminology; but the point of importance is that these tumours of the root contain a large amount of fibrous tissue, which contracts considerably, and thus produces compression of the bronchi primarily, and subsequently of the vessels and lymphatics at the root.

The root of the lung may be affected, of course, by extension from a primary growth in the mediastinum, but there is, as a rule, no difficulty in distinguishing the cases of primary root carcinoma from those secondary to mediastinal tumour, for with root cancer the mediastinum is affected but little, if at all.

The pathological appearances are striking. The lung on the affected side is usually somewhat smaller than the other. If a section be made through the lung, from the surface to the root, a whitish mass is seen radiating some distance from the root into the lung, surrounding the bronchi and following their divisions. It is rarely of any great thickness, even at the root, and may not measure as much as half an inch, and this rapidly tapers off as the bronchi divide, until, at a distance of two or three inches from the root, it may not be visible as more than a narrow band. It is hard on section, and cuts like fibrous tissue. In the centre of it are embedded the bronchial tubes, which are often considerably compressed, and sometimes almost completely obliterated. The radiating arrangement of the growth is very striking, but the actual amount of growth is really small, so that the growth may be easily overlooked, and the lesion missed, if not searched for.

The tubes may be empty, but frequently contain pus. It is unusual for them to present any lesion of their inner surface, but now and then the new-growth perforates the walls. It may then appear in the lumen as an ulcer, or as a papillary growth.

In the case of a primary cancer of the bronchus, which has invaded the peribronchial tissue, the growth in the bronchus is evident, either as a tumour or as an ulcer, and when it has extended outside, it does not spread into the lung in the same radiating fashion, but remains more as a localised tumour.

Because of the compression of the air-tubes, vessels and lymphatics, secondary changes in the lungs are more common with these forms of root-tumour than with those of the body of the lung.

Of 16 cases of primary root cancer, 12 occurred in men and 4 in women. Both sides were affected equally, *i.e.*, the right in 8 instances and the left in 8. The age was in all cases above 35, and the cases were distributed in age periods as follows:—

Total.	—40	—45	—50	—55	—60	—65	—70, etc.
16	2	3	1	1	2	5	2

[1] Wolf, *Fortschr. d. Med.*, vol. xiii. 725.

Root cancer of the lung, with secondary involvement of the pleura, showing—1. The mass of cancer (Scirrhus) at the root, compressing and almost obliterating the main bronchi. 2. The radiating, fan-like processes by which the growth extends from the root into the body of the lung, along the main bronchi. 3. The secondary changes in the lung, viz., the general œdematous consolidation of the whole lung, especially marked in the lowest parts, where two large necrotic cavities have been formed. 4. The massive thickening of the pleura, which at the base of the lung is 1½ inches in thickness. In most cases of cancer of the root of the lung, the new-growth is limited to the root, and does not involve the pleura at all. In this respect the specimen is peculiar.

Effects of the New-Growth on the Lung.—Primary tumours of the lung often remain localised in the lung. They may thus grow to a considerable size, and sometimes involve the whole lung. If they spread beyond the lung they may invade the mediastinum or adjacent parts, or they may involve the neighbouring lymphatic glands round the bronchi and trachea, or in the neck, and thus enlargement of the cervical glands above the clavicle is not uncommon.

Sometimes a primary growth in the lung may perforate a bronchus, and form a papillary or fungating mass within it, which may spread some distance within it. Portions of the tumour may then become detached and be coughed up. In one case[1] an actual cast of the tube, composed of the new-growth, was expectorated. Or particles may be detached and sucked into other bronchi, and there form centres of inflammation or of fresh growth.

There is an instance recorded[2] in which the pulmonary vein was invaded, and a small papillary tumour found within it; and another, in which the growth made its way into an old cavity, and presented as a villous or fungating growth within it.[3]

Lastly, a primary growth in the lung may become disseminated, and lead to secondary growths, most commonly in the lung itself, but sometimes in other parts.

It is, however, the secondary changes in the lungs themselves and in the pleura that are of chief interest.

Where there is a large mass in the lung, the parts around may be emphysematous, but more commonly they are somewhat collapsed, especially in the parts immediately round the growth which have not yet been invaded.

If the bronchi, blood vessels, and lymphatics are compressed by the growth, considerable changes are likely to be produced in the parts of the lung corresponding with the obstructed vessels.

Such obstruction is most likely to occur when the growth is at the root.

Fig. 178.

New-growth (cancer) of the lung, showing the way in which the tumour compresses the adjacent alveoli, before they become involved in the tumour. This was a section of a metastatic growth.

If the obstruction to the bronchial tubes be considerable, so that the air cannot freely enter the lung, the corresponding portion of the lung becomes collapsed, and subsequently œdematous. If the blood- and lymphatic-vessels, as

[1] Peacock, *Path. Soc. Trans.*, vol. xi. p. 65. [2] Bristowe, *Path. Soc. Trans.*, xi. 25.
[3] Kidd, *St. Barth. Hosp. Rep.*, xix. 227. Wolf, *l.c.*

well as the air-tubes, be compressed, the lung passes into the condition described as "solid œdema," *i.e.*, it is airless, and the vessels are filled with exudation partly serous and partly cellular.

In this a subacute inflammation may develop, and thus patches of consolidation, like broncho-pneumonia, be produced. After a time the central portions of these consolidated areas may degenerate, necrose, or even gangrene. These degenerating patches are often described as softened secondary growths. Cancer of the lung, however, does not usually soften and break down; and most of the softened masses described are simply areas of inflammation which have undergone necrotic degeneration. If, by communication with an unobstructed bronchus, the degenerate products can be discharged, a cavity results. The size of such cavities is sometimes considerable. Rösenberg describes one as large as a "child's head."

Cavities in the lung in connection with new-growth have three sources—(1) The necrosis of degenerate inflammatory tissue; (2) the softening or breaking down of the new-growth; (3) they may be of tubercular origin and antecedent to the new-growth. The occurrence of cancer in the course of chronic phthisis is not altogether rare, for there is no incompatibility between cancer and tubercle, as was once stated.[1]

When the new-growth reaches the pleural surface, inflammation of the pleura is likely to follow. This may lead to adhesions, through which the cancer may spread to the walls of the thorax. Often it leads to effusion. The fluid is usually serous in character and blood-stained. It may be purulent, but this is rare. Lastly, the pleura may be perforated and pneumothorax occur; this is, perhaps, the rarest complication, and I have only found one instance of it recorded.[2]

Primary Cancer of the Main Bronchi.

In connection with primary carcinoma of the root of the lung, primary cancer of the bronchi should be mentioned also, for the clinical symptoms are in many respects similar.

As already stated, primary cancer of the root develops round the bronchi, and not from a primary tumour or ulcer in the bronchi.

Primary bronchial cancer is very rare. Of 11 cases in which the bronchi were involved, in 2 certainly, and in 3 others in all probability, the affection of the bronchi was secondary to cancer outside it.

Thus only 6 cases of primary bronchial cancer are left. Of these, 1 presented simply a tumour in the lumen, and 1 an ulcer, without other changes. The other 4 had secondary changes also.[3]

1. A man of 70: the affected lung contained several secondary growths.
2. A man of 55: the tumour occupied the cavity of the right main bronchus; the glands surrounding the root were enlarged; the bronchi of the lung were filled with pus, and there were several patches of broncho-pneumonia in the upper lobe; there were secondary nodules in the liver and abdominal glands, and two small secondary growths in the upper lobe of the left lung.
3. A man of 58: the right main bronchus contained a cauliflower growth, which also filled the bronchi of the middle and lower lobes; the growth invaded the neighbouring bronchial lymphatic glands, and formed a small papillary tumour in the right pulmonary vein; secondary growths were found in the left lung, as well as in the dura mater, and some other parts.
4. A man of 42: the growth started at the bifurcation of the trachea, and spread along both bronchi; a cavity had formed at the left base, which had opened into the pleura. One secondary growth was found in the brain.

[1] Wolf, *l.c.* [2] *Ibid.* [3] *Ibid.*

The growths are described as squamous-celled or cylindrical-celled carcinoma, in about an equal number of cases, and they are supposed to arise from the mucous glands or the epithelium of the tubes.

These cases of bronchial cancer appear to be of short duration, and to terminate early, as the result of the secondary mischief they usually produce in the lungs.

Cancer of the Pleura.

Cancer of the pleura is in most instances secondary, and the result of direct extension from some cancerous growth in the parts near it. Usually adhesion takes place between its surfaces, and the growth spreads directly through the pleura to the parts beyond. These cases call for no comment.

At other times the growth in the pleura plays a more independent rôle.

The growths may occur as small, lenticular patches, about $\frac{1}{2}$–1 line in diameter, of the thickness of cardboard. They are occasionally in considerable number, and irregularly scattered. The condition closely resembles that met with in some forms of tubercle of the pleura.

These masses may become confluent, and so form patches of larger size, the "plaques circuses" of Cruveilhier.

The patches are usually thicker in the centre, but may be thickest at the periphery, where they are extending.

This is the usual form in which cancer of the pleura occurs, when it is associated with secondary disseminated nodules in the lungs.

A very considerable extent of the pleura, or even the whole of it, may become involved in this way, and when the visceral pleura is the chief seat of disease, it may form a sort of capsule to the lung, which in places may be of considerable thickness, even an inch or more. Extensive as the disease may then be, it may still be limited to the pleura and not involve the lung at all, except so far as it extends along the interlobar septa.

In another interesting form the cancer may develop tumours which project into the pleural cavity. These are commonest on the parietal or diaphragmatic surface. They are for the most part sessile, *i.e.*, have a broad base with rounded or nodular surfaces, and they may reach the size of a filbert or a small orange. At other times they may be pedunculated, and in a few instances have formed enormous tumours.

Wilkes[1] describes a case in a man of 30, following a primary growth in the fibula. Three or four secondary nodules of small size were found in each pleura, and one on the right side as large as the heart, growing from the surface of the visceral pleura, and pendulous.

In some instances the growth, though secondary, undergoes the most extraordinary development in the pleura; this is most frequently seen in the costal pleura, subsequent to cancer of the breast, or in the diaphragmatic pleura, secondarily to cancer of the peritoneum. In the latter case, the growths may extend through the diaphragm, apparently without involving the muscular tissue.

Thus in a case at St. Bartholomew's Hospital,[2] the cancer was primary in the peritoneum, both surfaces of the diaphragm were thickened with new-growths, and from each surface there were pendulous nodular masses, as well as similar nodular masses on the under surface of both lungs connected with the visceral pleura.

In another case, under my own care, at the Chest Hospital, Victoria Park, in which the left breast had been removed for cancer two years previously, the patient came under observation for chest symptoms and died. On *post-mortem* examination the whole mediastinum was found

[1] *Path. Soc. Trans.*, vol. x. p. 244.
[2] *Post-mortem Reg. St. Barth. Hosp.*, vol. xx. p. 384.

invaded by cancer, which had spread from the pleura just below the amputated breast, and from the mediastinum the growth had spread to the pericardium and to both pleuræ. On the right side the whole pleura was involved—parietal, costal, visceral and diaphragmatic; so that the lung was enveloped in a thick capsule, measuring, at the root of the lung and at the diaphragm, more than an inch in thickness.

The pleural cavity was completely obliterated, except for a space at the base, which held a pint of blood-stained fluid.

The left pleura was affected similarly, but only on the visceral surface near the root. From the chest the growth had spread to the peritoneum through the diaphragm, but without apparently involving the muscular substance, though the peritoneal surface, like the pleural, was covered with a thickened layer of new-growth; the capsule of the liver was similarly involved, and so, more or less, was the whole peritoneum.

The new-growths were confined to the serous membrane; no secondary nodules were found either in the liver or in the lung, or in any other organ.

Secondary growths, however, may affect the visceral pleura, and be confined to it, where the primary growth has been in some distant part, without any direct connection being evident, *i.e.*, metastasis may be confined to the pleura, as in the following case.

The new-growth was in the lower part of the peritoneum; the only seat of secondary growth was in the visceral pleura of the lung. The diaphragm was free, as well as the parietal pleura of both lungs; at the base, however, the visceral pleura was covered with numerous secondary nodules on both sides.

Primary Cancer of the Pleura.

Cancer of the pleura may be primary. It is a very rare affection, and I have only been able to find 12 cases recorded.

In 11 of these, 7 occurred in men and 4 in women; the right side was affected in 6, the left in 3, and both in 2.

The ages were as follows:—10 years, 37 (2 cases), 43, 44, 46, 54, 58 (2 cases), 61, and 69.

The growth is variously described as "columnar-celled carcinoma," "cylindrical-celled carcinoma," or "epithelial cancer."

It is characterised in all cases alike by the amount of fibrous tissue which it contains, so that it cuts almost like a piece of tendon.

The following is a brief abstract of the recorded cases.

1. M., 54.[1] Left pleura. Serous effusion. Tapped sixteen times; often 70 to 90 ounces at a time. Empyema. Rib resected. Malignant disease found. Death shortly after. Duration, eighteen months. *Post-mortem*, very extensive infiltration of the whole left pleura, costal, visceral, and diaphragmatic. Growth extending also between lobes. Cylindrical-celled carcinoma.

2. F., 58.[2] Similar growth right side. Duration, fourteen months. Tapped twice, but little fluid obtained. *Post-mortem*, right pleura very thick, from 1 to 2 inches. Interlobular tissue also involved, $\frac{1}{8}$ to $\frac{1}{4}$ of an inch thick. Lung completely encased. In one place only growth extended as a small nodule into lung. Cylindrical-celled epithelioma.

3. M. 37.[3] Pleural effusion. In the last two weeks of life hæmoptysis almost daily, sometimes in a considerable amount. Duration, ten months. *Post-mortem*, pleura $\frac{1}{4}$ to $\frac{3}{8}$ of an inch thick; growth extending also from the root into the lung and between the lobes; similar growths in the liver and the glands above the clavicle; some inflammatory changes in the lung, with small cavities. "Squamous epithelioma."

This was a mixed case, but the growth was probably primary in the pleura.

4. F., 58.[4] Pain in the side. Dyspnœa. Dry cough for fifteen months. *Post-mortem*, numerous hard nodules in the right pleura and on the diaphragmatic surface; a few also in the lung; scirrhus.

5. M., 64.[5] Ailing a few weeks only, possibly seven; admitted with right pleural effusion. Death from dyspnœa. *Post-mortem*, right pleura filled with 140 ounces of blood-stained fluid; numerous growths the size of beans or peas, confined to the parietal pleura and diaphragm; cancer.

[1] Benda, *Deut. med. Woch.*, May 20, 1897.
[2] Thomas Harris, *Jour. of Pathol.*, vol. ii.
[3] *Ibid.*
[4] Mayne, *Dubl. Hosp. Gaz.*, 1857, p. 21.
[5] Coats, *Glasg. Med. Jour.*, 1889, p. 19.

6. F., 46.[1] Ailing for two or three years. Cough and dyspnœa for two months. Hæmorrhagic pleural effusion on the right side. On paracentesis, fluid contained numerous epithelial cells, from which the diagnosis was made of cancer. *Post-mortem*, right pleura contained 50 to 60 ounces of blood-stained fluid; right pleura an inch thick in places, white, tough, like fibrous tissue; right half of the diaphragm infiltrated; nodular growths on the peritoneal surface of the diaphragm, and also on the capsule of the liver. Secondary growths in the peritoneum, and also a few in the left pleura, the size of a pea or so; one nodular mass of small size in the lung on right side. Tumour, composed of dense, fibrous tissue, with columns of epithelial cells.

7. F., 61.[2] For one year, cough, dyspnœa, pain in the left side, and loss of flesh. Left arm weak recently. *Post-mortem*, left lung collapsed; pleura containing 26 ounces of blood-stained fluid; the left costal pleura $\frac{1}{16}$ of an inch thick, on the posterior surface $\frac{1}{4}$ of an inch thick; dense infiltration of the diaphragm $\frac{1}{3}$ to $\frac{1}{2}$ of an inch thick; secondary growths in superclavicular glands along the œsophagus, the bifurcation of trachea, and the arch of aorta; nodules on the parietal and visceral pericardium, and a few also in the liver and kidneys. Cylindrical-celled epithelioma.

8. M., 43.[3] Illness commenced with dysphagia eight months before death; rapid loss of flesh; large right-sided effusion; blood-stained fluid. *Post-mortem*, new-growth of the lower part of the right pleura and diaphragm, extending a little on to the left; nodules on the abdominal surface of the diaphragm; glands of the posterior mediastinum and of the abdomen a little enlarged and colloid. Epithelial cancer.

9. M.[4] Hæmorrhagic effusion. *Post-mortem*, primary medullary cancer of the right pleura, with some secondary nodules in right lung and in the liver.

10. M., 37.[5] Obstinate pleural effusion; paracentesis showed a few epithelial cells; case thought to be one of new-growth. *Post-mortem*, diffuse infiltration in patches and in confluent masses half an inch thick; lung compressed by fluid, but free from tumour except for some small nodules on section, which had developed from strands following the lymphatics; left pleura slightly affected. Endothelial cancer.

10*a*.[6] A similar case, a museum specimen, described by the same author; he calls both the cases Endothelial carcinoma, or *syn.* Pleuritis carcinosa or Lymphangitis carcinomatodes.

11. M., 44.[7] Eight months losing flesh and strength, with pain in the side; came under observation with left pleural effusion. Polymorphic epithelial cells found in fluid, which was blood-stained. Diagnosis made of new-growth. *Post-mortem*, left pleura uniformly thickened $\frac{1}{4}$ of an inch; no tumours; growth looked like connective tissue; no tubercle bacilli; endothelial cancer; metastasic growths connected with the lymphatics of the bronchi, mesentery, and retro-peritoneum.

The author considers that these cases are often confused with fibrous or callous pleurisy.

The disease has also been called Lymphangitis carcinomatodes, or, better, Lymphangitis prolifera.

12. F., 10.[8] Right pleura very thick; cavity obliterated, except for a space at the base holding 3 or 4 ounces; lung collapsed; new-growth spreading along interlobar and interlobular septa; a few discrete nodules on the left pleura. Carcinoma.

SYMPTOMS AND PHYSICAL SIGNS.—Of the symptoms and physical signs of new-growth of the lung and pleura we can hardly speak precisely, for none are pathognomonic or constant, and all may be absent.

Practically we may limit our consideration to malignant disease, *i.e.*, to sarcoma and cancer, for the innocent tumours are very rare, and, for the most part, give no evidence of their presence.

Symptoms.—The chief symptoms are dyspnœa and pain in the chest. Cough is common, but expectoration accidental.

When the growth is malignant, the history of the case, the loss of flesh and strength, and the general cachexia, may assist the diagnosis.

The variability in the symptoms and physical signs is to be explained by the position of the growths, their size, and their number.

[1] *Path. Soc. Trans.*, vol. xliv. 5.
[2] Pitt, *Path. Soc. Trans.*, vol. xxxix., p. 56.
[3] Collier, *Lancet*, 1885, No. 21.
[4] Kanders, *Wien med. Bl.*, 1880, Nos. 18 and 22.
[5] Neelsen, *D. Arch. f. kl. Med.*, xxxi. (1882), 375.
[6] *Ibid.*
[7] Fraenkel, *Verh. d. Congr. f. Inn. Med.*, 1892, p. 374.
[8] Lepine, *Bull. d. l. Soc. Anatom.*, 1869.

Onset.—In many cases the symptoms develop gradually, but in some instances the onset is sudden, the symptom being due to the rapid development of some complication.

Thus a man of 39[1] was in fair health till he was seized with sudden illness like a pneumonia. He had much fever, lost flesh rapidly, and sweated much at night.

Six weeks later he was admitted with signs of pleural effusion, it was thought, probably purulent. After an exploratory puncture an incision was made, and 8 ounces of pus evacuated.

The diagnosis of malignant disease was made ; little relief was experienced. The patient died of exhaustion three months later, four and a half months in all from the commencement of his illness.

The incision led into a cavity in the base of the lung. There was also another of smaller size near it. The malignant disease existed at the root of the lung, and extended for some distance into it.

Another case of the same kind, in a man of 62, is recorded in the same paper.

Dyspnœa.—In the body of the lung there may be a large growth, or several smaller ones, without the breath being affected at all.

This depends not so much upon the size of the tumours as upon their slowness of growth, the lungs having had time to adjust themselves gradually to the altered conditions.

Thus, in the case of osteo-sarcoma described, although the tumours were very large and in both lungs, symptoms were entirely absent until four weeks before death, and then the dyspnœa was chiefly due to a pleuritic effusion which developed rapidly.

When the tumours are of rapid growth, dyspnœa is hardly likely to be absent. Again, if the tumours are very numerous, though small, the shortness of breath may be considerable, and so greatly in excess of the apparent mischief in the lung as of itself to excite suspicion. This, as would be expected, was the case in those rare instances of carcinosis which, in general features, closely resemble disseminated tuberculosis of the lung.

A sense of pressure or compression in the chest is occasionally complained of. It generally stands in relation with the dyspnœa.

Pain.—Pain is not likely to be present unless the pleura be involved, or the nerves pressed upon in some part of the chest. Where the intercostal nerves are interfered with, the pain may be referred to distant parts ; and in one case, which I have described, the earliest symptom was intercosto-humeral neuralgia—pain, that is to say, down the inner side of the arm as far as the elbow.

Cough.— Cough is frequently absent. It varies with the amount of secretion in the air-tube and the difficulty of its expectoration. It may be paroxysmal, but it is not often a troublesome symptom. When complications occur, the symptoms due to them are introduced, and are likely either to mask completely the signs of tumour, or greatly increase the difficulties of diagnosis.

Decubitus.—This is one of the most suggestive symptoms. The patient finds that some position or another either reduces pain or difficulty in breathing, or greatly aggravates them, and so adopts one which becomes more or less constant throughout the case.

The decubitus varies greatly in different cases. Sometimes it is the affected side the patient avoids, sometimes the opposite one ; more often the patient is only comfortable when lying upon the back with the shoulders raised.

Of course the presence of a pleuritic effusion will largely determine the position preferred ; but in the absence of such complication, the explanation lies in the relation in which the tumour stands to the root of the lung and the

[1] *Path. Soc. Trans.*, xxxv. 84.

diaphragm, the patient finding that position most comfortable which most relieves those parts of pressure.

Physical Signs.—The physical signs vary according as the tumour is deep-seated or near the surface, the pleura involved, or secondary changes in the lungs present. If the pleura be involved, the usual signs of an affection of the pleura will be present, viz., dulness to percussion, and diminution or absence of the voice- and breath-sounds.

If the pleura be not affected, and the tumour deep-seated, there may be no physical signs at all so long as the surrounding lung-tissue contains air.

If the tumour be close beneath the chest-walls, or the lung consolidated, the percussion will be impaired; the voice- and breath-sounds may be increased and altered; but owing to the obstruction which the tumour causes to the entrance of air into the affected part, they are more often diminished or absent, and then the diagnosis from affections of the pleura becomes very difficult.

On inspection, often no changes in shape or movement are to be detected; but if the tumour be large, or there be a large pleuritic effusion, the affected side may be distended. On the other hand, the side may be smaller if the tumour be at the root of the lung and the lung collapsed.

If there be any change in the shape, whether by way of increase or decrease, the respiratory movements will be diminished.

If the tumours in the lung be numerous and of small size, the physical signs may resemble those of disseminated broncho-pneumonia, and as, in the adult, this is usually of tubercular origin, such a case is likely to be diagnosed as phthisis, especially if, as frequently happens, the temperature be raised. Thus in Fagge's case of carcinosis, the only diagnosis made during life was that of bronchitis and broncho-pneumonia.

It is when the primary tumour is at the root of the lung that the physical signs become most puzzling. Here the side may be contracted, and the breath- and voice-sounds diminished, or even completely absent. So long as no secondary changes occur in the peripheral parts of the lung, the percussion may not be affected; but when the lung has become œdematous or consolidated, the percussion will become dull, and the physical signs be the same as those of pleuritic effusion. If, as usual, the physical signs be most marked at the base, and at the same time the temperature be raised, the case will present the features of a localised empyema.

The *temperature* is not raised with tumours unless there be some inflammatory complication, *e.g.*, consolidation with degeneration of the lung, or pleurisy. It is the elevation of temperature which introduces many of the difficulties into the diagnosis, for the case may then resemble empyema, phthisis, or subacute pneumonia.

Expectoration is not constant. Indeed, it is often absent until the new-growth has involved the actual walls of the air-tubes. It may be absent even when perforation has taken place, owing to the obstruction to the air-tubes, unless the perforation be on the tracheal side of the tumour. The sputum may be catarrhal in character, *i.e.*, mucopurulent, hæmorrhagic, or contain some of the new-growth or shreds of lung tissue. *Hæmoptysis* in any quantity is not common, unless the tumour has made its way into one of the large air-tubes. Usually it amounts to little more than streaks of blood upon the surface of the mucopurulent sputum, as in chronic bronchitis. It is rarely abundant, but it has in some cases been fatal.

Walsh states that, in cases of cancer of the lung, less than one-third escape hæmoptysis altogether.

Newton Pitt[1] records a case in which several ounces of blood-stained expectoration, on some days almost pure blood, were brought up daily for some months, the patient ultimately dying of exhaustion.

Two cases of fatal hæmoptysis, with cancer of the lung, are recorded by Walsh[2] and Hoyle.[3]

In two cases the sputum was of a peculiar olive or grass-green colour. This is obviously accidental and of no significance, for it is rare with tumour of the lung, and has been met with in other affections, *e.g.*, phthisis and pneumonia.

The sputum may also contain particles of lung tissue, or of the growth itself, when a bronchus has been perforated.

In a case of sarcoma of the lung under my own observation, all the tubes of the affected lung were filled with a gelatinous substance of the same nature and appearance as the original tumour, and the patient brought up this subtance in considerable quantity during life.

Occasionally the growth is more coherent, so that definite particles of tumour may be brought up.[4] In a most remarkable case recorded by Peacock,[5] an actual cast formed of new-growth was expectorated. But even when the growth has made its way into the air-tubes, nothing characteristic may be brought up, owing to the occlusion of the main tubes by the original tumour.

The superficial *lymphatic glands* sometimes become enlarged.

The group of glands most likely to be affected is at the root of the neck, above the clavicle, but this enlargement is more often absent than present. Enlarged glands have been also met with in the axilla; rarely with simple intra-thoracic tumours, usually only where the walls of the thorax are involved.

If the tumour, whether in the pleura or the lung, lead to pressure upon the intra-thoracic veins, the *superficial veins* over the corresponding part of the thorax may be dilated. The nerves may be involved in the same way and pain caused, which may be limited to the seat of pressure or referred to some distant points in the course of the nerve.

Clubbing of the fingers is stated to occur occasionally by Lebert, but I have not seen it myself, nor do I know of any recorded case to refer to.

DIAGNOSIS.—Many of the difficulties of diagnosis have already been incidentally referred to in discussing the physical signs.

Speaking generally, chest symptoms or physical signs of any kind occurring in a patient, who is already the subject of malignant disease elsewhere, are in most cases correctly referred to secondary growths within the thorax.

When the seat of primary growth is within the thorax, the diagnosis is more difficult; but the age, the cachexia, and the history of rapid loss of flesh and strength may suffice to indicate the diagnosis of malignant disease, even when the physical signs and pulmonary symptoms are not sufficient actually to locate it; still more if the physical signs and symptoms are pulmonary, unilateral, and anomalous.

The difficulties of diagnosis are twofold: first, the prime disease may be masked by the complications to which it has given rise; secondly, in the absence of complications, the symptoms and physical signs may admit of other and more probable interpretations.

[1] *Guy's Hosp. Rep.*, xxxiv. [2] *Loc. cit.*, and *Jour. of Anat. and Phys.*, July 1883.

[3] Elliott, *Brit. Med. Jour.*, April 25, 1874. Janssen, *Corr. bl. f. Schweizer. Aerzte*, 1885, No. 23.

[4] *Cf.* Humpel's case, *Petersburg Med. Woch.*, 1876, No. 40.

[5] *Path. Soc. Trans.*, xi. 65.

1. *Complications on the side of the pleura, e.g.*, pleuritic thickening or pleuritic effusion.

The physical signs and symptoms are those of affection of the pleura, and the difficulty consists in determining the cause of the pleurisy.

This question is sometimes settled by the history, as in the case of osteo-sarcoma already described, or by some peculiarities in the pleurisy itself. Thus double pleurisy is always suspicious, for it probably depends upon a lesion which can involve both sides, *e.g.*, tubercle; if not tubercle, it is probably new-growth.

A hæmorrhagic effusion is also suspicious, but it is not conclusive; for in many cases of cancer the effusion is serous and not hæmorrhagic, and when the effusion is hæmorrhagic, it may equally well be due to tubercle as to new-growth. The quantity of blood in such effusions varies greatly. There may not be more than enough to give it a pink colour, or there may be sufficient to make it look almost like pure blood. Strange to say, it is often bright in colour, as if the blood had not been long mixed with the serum; but as this cannot usually be the case, we must conclude that blood in the pleura does not usually undergo the colour-changes so readily as it does in other parts of the body. The fluid, when drawn from the chest, may, however, be dark in colour, or even almost black. Thus I have withdrawn an almost black serous fluid on several occasions from the pleura of an elderly man affected with malignant disease of the lung or pleura, secondary to malignant disease of the stomach.

Empyema may occur with cancer, but it is rare.

2. The *complications in the lung* often cause still greater difficulties of diagnosis, viz., œdema, inflammatory consolidation, and breaking down.

Owing to the obstruction to the passage of air through the tubes, the voice- and breath-sounds are deficient, and the signs are suggestive of effusion, from which the diagnosis is often impossible except by the needle. Even then the conclusion drawn may be wrong, as in a case I have quoted, where the general conditions seemed to point to an empyema, and when the needle was inserted, pus was withdrawn; an incision was made, but only a drachm or two of pus obtained.

The *post-mortem* showed that there was no empyema; that the pus had come from a cavity in the base of the lung, the result of secondary changes produced by the new-growth at the root.

The rise of temperature associated with many of these complications greatly complicates the diagnosis, for it may lead to the suspicion of empyema (as in the case just mentioned) or of phthisis.

In respect of phthisis, due consideration of the age of the patient, of the history and general character of the disease, often enables the correct diagnosis to be made. The examination of the sputum may confirm the conclusion arrived at, by the discovery, in the one case, of the tubercle bacillus, and in the other of portions of growth; but in many cases the diagnosis is a matter of conjecture only, and remains uncertain until the *post-mortem* examination.

DURATION.—The average duration of malignant disease of the lung is short. In the case of cancer, secondary growths within the thorax are among the latest manifestations of the disease, and life is rarely prolonged beyond a few weeks.

In secondary sarcoma the disease sometimes runs a very rapid course and sometimes a very slow one. Thus death may occur within a very few weeks of the first onset of symptoms. In other cases the symptoms may not become serious for a considerable time, as in the cases quoted, in which two and a half years in one case and four years in another elapsed from the removal of the primary tumour.

The duration of primary cancer of the lung and pleura is also short, but is difficult to determine, owing to the indefinite symptoms which it produces in the early stage.

A few months—six to eight—appears to be the average duration; something, at any rate, considerably under a year. Possibly in the multiple disseminated form (carcinosis) the disease may run its course in a few weeks. With tumour of the root of the lung, in which the cancer is of the fibrous type and limited to one side, it is quite possible that the duration may be longer, and in some of the cases a history of two or three years' illness is given.

CAUSE OF DEATH.—Death is usually the result of asthenia. Some patients die from hæmoptysis, but this is really rare, and some from other complications rarer still, such as pneumothorax or gangrene of the lung.

Next to asthenia, the commonest cause of death is suffocation, but this need not be due directly to the tumour, but rather to the complications which have been produced in the pleura or in the lung. The onset of grave symptoms, such as extreme dyspnœa and cyanosis, may be very sudden; for it is evident that where a large part of the lung is already occupied by growth, slight changes in the remainder, such as may be the result of slight congestion, or of the collapse produced by even a small effusion, will suffice to give rise to urgent symptoms.

TREATMENT.—The treatment can, of course, be only symptomatic, for there is nothing at present known which has the power of controlling the growth of tumours. Surgical interference is obviously impracticable for many reasons, not the least of which is the uncertainty of diagnosis.

THE END.

INDEX.

Abductor paralysis, 65.
,, ,, course of, 68.
,, ,, diagnosis of, 69.
,, ,, duration of, 68.
,, ,, etiology of, 68.
,, ,, forms of, arthritic, 67.
,, ,, ,, cicatricial, 67.
,, ,, ,, myopathic, 67.
,, ,, ,, neuropathic, 67.
,, ,, history of, 69.
,, ,, treatment of, 69.
Abscess of chest wall, suggesting empyema, 718.
,, of lung, 362.
,, ,, chronic, 364.
,, ,, following acute inflammation, 363.
,, ,, ,, pneumonia, 259.
,, ,, forms of, 363.
,, ,, physical signs of, 364.
,, ,, prognosis of, 365.
,, ,, symptoms of, 364.
,, ,, treatment of, 366.
,, in mediastinum, 106.
Actinomycosis of lung (and pleura), 841.
,, ,, amyloid disease in, 848.
,, ,, analysis of cases of, 847.
,, ,, course of, 849.
,, ,, death, causes of, in, 851.
,, ,, diagnosis of, 851.
,, ,, duration of, 850.
,, ,, history of, 852.
,, ,, involving chest walls, 848.
,, ,, metastasis in, 849.
,, ,, miliary dissemination in, 848.
,, ,, pathology of, 842.
,, ,, physical signs of, 850.
,, ,, primary, 846.
,, ,, prognosis in, 851.
,, ,, pyæmic form of, 845, 849.
,, ,, secondary, 846.
,, ,, treatment of, 851.
Addison's disease in relation to phthisis, 522.
Adductor spasm of the larynx, 56.
Adhesions, pleural, 753.
Aërocele, 99.
Ægophony, 660.
Africa, South, for the phthisical, 561.
Aged, bronchitis of the, 155.
Ague, bronchitis in, 183.
Air, composition of, in pneumothorax, 794.
Air-containing tumours of neck, 100.
Air-embolism, 359.
Air-passages, anatomy of, 3.
Air-tubes, stricture of, 87.
,, syphilis of, 84.
,, tumours of, 91.
Alar chest, 453.
Albuminous expectoration, 162, 691.
,, ,, theories of, 693.
Albuminuria in phthisis, 486.
,, pneumonia, 285.
Alcoholism, bronchitis in, 183.
,, in relation to phthisis, 521.
,, ,, pneumonia, 297.
Algiers for the phthisical, 560.
Alveoli of lung, anatomy of, 6.
America, Western, for the phthisical, 561.
,, South, for the phthisical, 562.
Amyloid disease in actinomycosis, 848.
,, ,, empyema, 711.
,, ,, phthisis, 511.
Aneurysm, abdominal, empyema resembling, 708.
,, of pulmonary artery, 382, 383, 387.
,, ,, clotting in, 384.
,, ,, pathogenesis of, 385.
,, thoracic, diagnosis from mediastinal tumour, 103.
,, ,, compressing trachea, 102.
,, ,, empyema resembling, 718, 722.
Angeio-neurotic œdema, 54.
Anthracosis, 197.
Antitoxin treatment of diphtheria, 50, 51.
,, ,, pneumonia, 253, 306.
Apex-pneumonia, 299.
Aphasia in pneumonia, 287.
Aphonia in phthisis, 508.
Apneumatosis, 225.
Apyretic phthisis, 458.
,, pneumonia, 265.
Arterial tension in pneumonia, 280.
Artery, pulmonary, blood pressure in, 239.
Aspergillosis of lung, 856.
,, ,, history of, 859.
Aspiration-pneumonia, 323.
Asthenic bronchitis, 155.
Asthma, 566.
,, age in relation to, 571.
,, bronchitis in, 184.
,, causes, exciting, of, 582.
,, ,, ,, diagram of, 583.
,, ,, predisposing, 571.
,, Curschmann's spirals, 569.
,, cyanosis in, 570.
,, diagnosis of, 587.

Vol. I. comprises pages 1–398. Vol. II. pages 399 to the end.

Asthma, duration of attack, 570.
,, etiology, 571.
,, forms of, 587.
,, hay, 577.
,, heredity in, 571.
,, history of, 598.
,, humidum, 159.
,, hysterical, 581.
,, Leyden's crystals in, 569.
,, locality, effect of, on, 599.
,, paroxysm, treatment of, 590.
,, ,, ,, by belladonna, 591.
,, ,, ,, caffein, 594.
,, ,, ,, chloroform, 592.
,, ,, ,, coffee, 594.
,, ,, ,, inhalations, 596.
,, ,, ,, ipecacuanha, 593.
,, ,, ,, nitre, 596.
,, ,, ,, nitrite of amyl, 594.
,, ,, ,, nitroglycerine, 594.
,, ,, ,, opium, 592.
,, ,, ,, pilocarpin, 593.
,, ,, ,, smoking, 595.
,, ,, ,, stramonium, 591.
,, predisposing causes of, 571.
,, prognosis in, 588.
,, prophylaxis in, 589.
,, relation of, to affections of heart, 574.
,, ,, ,, kidney, 575.
,, ,, ,, lungs, 574.
,, ,, ,, mediastinum, 578.
,, ,, ,, neck, 578.
,, ,, ,, nervous system, 580.
,, ,, ,, nose, 576.
,, ,, ,, pharynx, 578.
,, ,, ,, stomach, 574.
,, ,, to gout, 575.
,, ,, insanity, 581.
,, ,, lead-poisoning, 575.
,, sex, influence of, in, 571.
,, sputum in, 569.
,, symptoms of, 567.
,, theories of, 584.
,, treatment of, general, 589.
,, ,, paroxysm, 590.
,, varieties of, 587.
Atelectasis, compression, 227.
,, congenital, 18, 226.
,, history of, 231.
,, obstruction, 228.
Atomization, treatment of bronchitis by, 135.
Australia for the phthisical, 561.

Bacteriology of actinomycosis, 841.
,, bronchitis, 114.
,, broncho-pneumonia, 347.
,, croupous pneumonia, 256.
,, diphtheria, 53.
,, empyema, 713.
,, gangrene of lung, 366.
Bacteriology of phthisis, 401.
,, pneumono-mycoses, 840.
Barrel-shaped chest, 207.
Basic empyema, 717.
,, phthisis, 414.
Bayle, gray granulation of, 409.
Bell-sound in pneumothorax, 787.
Bilharzia hæmatobia in lung, 860.
Bleeding in bronchitis, 141.
,, congestion of lung, 238.
,, emphysema, 217.
Blood, changes in, in phthisis, 487.
,, ,, pneumonia, 281.
Blood-casts of bronchi, 376, 464.
Blood-pressure in pulmonary artery, 239.
Blood-spitting (see *hæmoptysis*), 375.
Blood-vessels of lungs, 6 to 9.
Bournemouth for the phthisical, 563.
Brain abscess after empyema, 709.
Breathing, Cheyne-Stokes, 618.
Breath-sounds, varieties of, 12.
Bright's disease, bronchitis in, 183.
,, in relation to pleurisy, 658.
Bronchi, anatomy of, 4.
,, blood-casts of, 376.
,, cancer of, 97, 884.
,, enchondroma, 97.
,, syphilis of, 86, 89.
,, tubercle of, 83.
,, tumours of, 97.
Bronchial-atresia in phthisis, 418.
,, breathing (general), 11.
,, ,, in pleuritic effusion, 665.
,, casts in phthisis, 464.
,, ,, plastic bronchitis, 167.
,, croup, 167.
,, glands in phthisis, 419.
,, ,, enlarged, acute, 105.
,, ,, ,, chronic, 104.
,, ,, ,, symptoms of, 105.
,, ,, ,, treatment of, 106.
,, ,, impacted in larynx, 106.
,, ,, suppurating, 106.
,, ,, new-growths of, 104.
,, polyp, 167.
Bronchiectasis—acute and chronic, 184.
,, broncho-pneumonia, causing, 329.
,, croupous pneumonia causing,
,, causes of, 189.
,, complications of, 191.
,, cylindrical, 185.
,, diagnosis of, 191.
,, phthisis associated with, 191, 418.
,, prognosis in, 192.
,, results of, 190.
,, saccular, 187.
,, seat of, 190.
,, symptoms of, 191.
,, trabecular, 188.
,, treatment of, 192, 545.
Bronchiolectasis, 186.
Bronchiolitis, 150.
Bronchitis, acute, of adult, 147.
,, ,, of aged, 155.

Bronchitis, acute, of children, 13.
,, age in, 110.
,, anatomy, pathological, of, 117, 121, 122.
,, asthenic, 155.
,, bacteriology of, 114.
,, capillary, of child, 150, 324.
,, ,, of adult, 154.
,, Cheyne-Stokes breathing in, 126.
,, chronic, 156.
,, collateral hyperæmia causing, 111.
,, complications of, 123.
,, congestion of lungs in, 115.
,, croupous, 167.
,, diagnosis of, 132.
,, dusts, inhalation of, causing, 113, 197.
,, etiology of, 108.
,, extension of inflammation causing, 111.
,, feeble health causing, 110.
,, fœtid, 163.
,, fogs causing, 113.
,, forms of, 146.
,, frequency of, 116.
,, general diseases, effect of, on, 111.
,, geographical distribution of, 109.
,, hæmoptysis in, 125.
,, heart, effect of, upon, 128.
,, ,, -failure in, 129.
,, history of, 108.
,, infectious diseases, effect of, 111.
,, inheritance, effect of, 114.
,, inspiratory recession in, 124.
,, local irritation causing, 112.
,, morbid anatomy of acute, 117.
,, ,, ,, cardiac, 122.
,, ,, ,, chronic, 121.
,, mortality of, 116.
,, pain on coughing in, 126.
,, pathological complications of, 123.
,, physical signs of, 123.
,, plastic, 167.
,, ,, lesions associated with, 169.
,, previous attacks, effect of, on, 111.
,, prognosis in, 132.
,, prophylaxis of, 145.
,, putrid, 163.
,, race, effect of, on, 110.
,, season, effect of, on, 109.
,, secondary, caused by collateral hyperæmia, 180.
,, ,, caused by mechanical congestion, 179.
,, ,, caused by morbus cordis, 179.
,, ,, influence of ague in, 183.
,, ,, ,, alcoholism, 183.
,, ,, ,, asthma, 184.
,, ,, ,, Bright's disease, 183.
,, ,, ,, fevers, 181.
,, ,, ,, general diseases, 182.
,, ,, ,, gout, 183.
,, ,, ,, phthisis, 514.
,, ,, ,, pneumonia, 293.
Bronchitis, secondary, influence of pregnancy in, 183.
,, ,, ,, rheumatism, 183.
,, ,, ,, syphilis, 183.
,, septic, 163.
,, sex in, 110.
,, sputum in, 124.
,, symptoms of, 125.
,, temperature in, 127.
,, treatment of, in general, 132.
,, ,, by alteratives, 143.
,, ,, astringents, 143.
,, ,, bleeding, 141.
,, ,, counter-irritation, 140.
,, ,, cupping, 141.
,, ,, demulcents, 139.
,, ,, derivatives, 139.
,, ,, diaphoretics, 141.
,, ,, diet, 144.
,, ,, emetics, 142.
,, ,, expectorants, 137.
,, ,, inhalation, 133.
,, ,, intratracheal injections, 137.
,, ,, narcotics, 142.
,, ,, sprays, 135.
,, ,, stimulants, 144.
,, ,, venesection, 141.
,, ,, water cures, 145.
,, venous congestion causing, 111.
Broncho-blennorrhœa, 159.
Bronchocele, aërial, 99.
,, common, 97.
,, exophthalmic, 97.
,, producing pressure on trachea, 97.
Broncho-pneumonia, 323.
,, acute congestive, 324.
,, bacteriology of, 347.
,, bronchiectasis after, 329.
,, etiology of, 330.
,, forms of, aspiration, 323.
,, ,, deglutition, 323.
,, ,, embolic, 323.
,, ,, metastatic, 323.
,, ,, primary, 340.
,, ,, secondary, 332.
,, ,, septic, 323.
,, ,, table of, 350.
,, history of, 350.
,, idiopathic, 324.
,, induration of lung after, 329.
,, in the adult, 339.
,, in little children, 326.
,, in hypostatic congestion, 232.
,, in phthisis, 514.
,, pathology of, 326.
,, primary, 324, 329, 330, 340.
,, ,, diagnosis between, and secondary, 340, 343.
,, results of, 329.
,, secondary, 324, 326, 330, 332.
,, ,, access of, 334.
,, ,, clinical course of, 332.
,, ,, diagnosis of, 337.
,, ,, duration of, 336.
,, ,, mortality of, 336.

Broncho-pneumonia, secondary, physical signs of, 332.
,, ,, prognosis in, 336.
,, ,, temperature in, 334, 341.
,, ,, treatment of, 338.
,, temperature in, 344.
Bronchorrhœa, 159.
,, serosa, 160, 243.
Brown induration of the lung, 233, 235.
Bruit d'airain, 787.
,, de pot fêlé in pleuritic effusion, 665.
,, ,, pneumonia, 277.
,, de fistule in pneumothorax, 789.
Bucco-pharyngeal tuberculosis in phthisis, 424.

Caffeine in asthma, 594.
Calcareous masses expectorated in phthisis, 465.
Canary Islands for the phthisical, 560.
Cancer, relation of, to phthisis, 517.
,, of the lung, 877.
,, ,, primary, of the main bronchi, 884.
,, ,, ,, of the root, 882.
,, ,, secondary, by extension, 878.
,, ,, ,, infection through air-tubes, 880.
,, ,, ,, ,, through blood vessels, 879.
,, ,, ,, ,, through lymphatics, 879.
,, of the pleura, 885.
,, ,, primary, 886.
Capillary bronchitis, 324.
,, in adult, 154.
,, in child, 150.
Carcinoma of lung, 882.
Carcinosis of the lung, 879, 881.
Cardiac murmurs in emphysema, 212.
,, pneumonia, 292.
Caries of bronchial cartilages, 70.
Carnified lung, 644.
Casts, bronchial, blood, 376, 464.
,, ,, fibrinous in phthisis, 464.
,, ,, ,, in plastic bronchitis, 167.
Caseating pneumonia, 407.
Caseation in phthisis, 400, 405.
Catarrh, pituitary, 160.
,, suffocative, 150, 324, 325.
Catarrhal croup, 35.
,, laryngitis, 32.
,, spasm, 35.
Catarrhe sec, 159.
Cavities in lung, 410, 455, 475.
,, ,, bronchi in relation to, 412.
,, ,, bronchiectatic, 411.
,, ,, changes in, 413.
,, ,, contents of, 412.
,, ,, diagnosis of large, from pneumothorax, 811.
,, ,, distribution of, 412.
,, ,, dry, 410.
,, ,, in children, 455.
Cavities in lung, signs of, 475.
,, ,, site of, 413.
,, ,, trabeculated, 411.
,, ,, vessels in relation to, 412.
Chalicosis, 198.
Charcot-Leyden's crystals, 160.
Chest, alar, 453.
,, barrel-shaped, 207.
,, flat, 453.
,, pterygoid, 453.
,, punctured wound of, rarely causing pneumothorax, 772.
Cheyne-Stokes breathing, 617.
,, ,, associated phenomena of, 621.
,, ,, diagnosis of, 623.
,, ,, in bronchitis, 126.
,, ,, in pneumonia, 287.
,, ,, theories of, 623.
Children, acute bronchitis in, 150.
,, apex-pneumonia, greater frequency of, in, 258.
,, capillary bronchitis in, 150.
,, catarrhal laryngitis in, 35.
,, cavities in lung in, 455.
,, laryngismus stridulus in, 58.
,, phthisis in, 454.
,, pneumonia in, 300.
,, spasm of larynx in, 58.
,, suffocative catarrh in, 150.
,, syphilis of larynx in, 85.
,, tuberculosis in, 429.
,, webs in larynx, congenital, 88.
Child's lung, peculiarities of, 332.
Chlorides in urine in pneumonia, 285.
Chronic excavation of lung, 502.
Chylothorax, forms of, 765.
,, analysis of fluid in, 765.
Cicatricial stricture of air-tubes, 87.
Cicatrization of lung in phthisis, 408.
Cirrhosis of liver in relation with phthisis, 426, 521.
,, of lung, 317, 409.
Classification of diseases of respiratory organs, 19.
Climate in relation to bronchitis, 144, 146.
,, ,, phthisis, 440, 554.
Clots in heart in pneumonia, 258, 292.
Clubbing of fingers, 488.
,, ,, disappearance of, 490.
,, ,, in healthy persons, 491.
,, ,, rapid development of, 490.
Coccidiosis of lung, 859.
Coffee in asthma, 594.
Cold bathing in pneumonia, 307.
,, packing in pneumonia, 307.
Collapse of lung, 225.
Collateral fluxion, 233, 238.
,, hyperæmia, bronchitis due to, 180.
Compensatory emphysema, 220.
Complementary hypertrophy of lung in phthisis, 418.
Congenital atelectasis, 18.
,, infantile stridor, 62.

Congestion of heart in failing heart, 129.
,, of lung, 233.
,, ,, acute inflammatory, 234.
,, ,, ,, in acute bronchitis, 234.
,, ,, ,, in high fever, 235.
,, ,, ,, preceding pneumonia, 279.
,, ,, bleeding in, 238.
,, ,, cardiac, 235.
,, ,, chronic, causing bronchitis, mechanical, 235.
,, ,, collateral fluxion causing, 238.
,, ,, hypostatic, 231, 240.
,, ,, obstruction to air-tubes, 238.
,, ,, in pulmonary capillaries, 238.
,, ,, pulmonary veins causing, 238.
Convulsions in whooping-cough, 606.
Cough, treatment of, in phthisis, 547.
Counter-irritation in bronchitis, 140.
,, ,, in pleurisy, 658.
,, ,, in pneumonia, 307.
Creasote in phthisis, 543.
Crepitation, 16.
,, fine hair, in pneumonia, 275.
Crisis in pneumonia, 265.
Critical days in pneumonia, 269.
,, discharges in pneumonia, 265.
Croaking, respiratory, in babies, 62.
Croup, bronchial, 167.
,, membranous, 39.
,, spasmodic, 35.
Croupous pneumonia, 245.
Crystals, Charcot-Leyden, 160.
Cupping in bronchitis, 142.
Curschmann's spirals, 160, 569.
Cyanosis in asthma, 570.
,, emphysema, 211.
,, membranous laryngitis, 43.
,, obstruction by foreign body, 26.
,, phthisis, 480.
,, whooping-cough, 601, 613.

Decubitus in cancer of lung, 888.
,, pleurisy, 656, 667.
,, pneumonia, 273.
Davos for the phthisical, 558.
Deglutition-pneumonia, 323.
Delirium in pneumonia, 286.
,, tremens, forms of, 305.
,, ,, in pneumonia, 286.
Desquamative pneumonia, 323, 407.
Diabetes mellitus in relation to phthisis, 523.
Diarrhœa in phthisis, 484.
,, ,, treatment of, 550.
Diet in bronchitis, 144.
,, hæmoptysis, 396.
,, phthisis, 552.
Dilatation murmurs, 129.
,, in emphysema, 212.
Diaphragm, affections of, 827.
,, ,, mechanical, 827.
,, ,, muscular, 827.
,, ,, nervous, 827.
Diaphragm, degeneration of, from pressure, 827.
,, hernia of, 831.
,, inflammation of, in connection with pleurisy, 709.
,, inhibition of, 828.
,, paralysis of, 836.
,, ,, hysterical, 828.
,, perforation of, by empyema, 709.
,, rupture of, diagnosis of pneumothorax from, 812.
,, spasm of, 828.
Diaphragmatic empyema, 717.
,, hernia, 831.
,, ,, congenital, 832.
,, ,, traumatic, 833.
,, pleurisy, 750.
,, ,, diagnosis of, 752.
,, ,, physical signs of, 751.
,, ,, results of, 752.
,, ,, symptoms of, 751.
,, ,, treatment of, 752.
Diphtheria, see *membranous laryngitis*.
Displacement of organs in pleuritic effusion, 778.
,, ,, pneumothorax, 778, 784.
,, of heart, 785, 786.
,, ,, cardiac murmurs from, 785.
,, of liver, 784.
Dissecting pneumonia, 315.
Dropsy of the pleura, 756.
Dry tapping, 669.
Duodenum, tubercular ulceration of, in phthisis, 423.
Dusts causing bronchitis, 113.
Dyspeptic phthisis, 483.
Dysphagia in phthisis, 484.

Egypt for the phthisical, 560.
Embolic pneumonia, 298, 323.
Embolism, air, 359.
,, cerebral, after empyema, 710.
,, fat, pulmonary, 354.
Emetics in bronchitis, 142.
Emphysema, 200.
,, age in, 205.
,, barrel-shaped chest in, 207.
,, bronchitis in relation to, 197.
,, cardiac murmurs in, 212.
,, chest-movements in, 208.
,, consequences of, 201.
,, cyanosis in, 211.
,, diagnosis of, 214.
,, dilatation of heart in, 212.
,, ,, murmurs in, 212.
,, forms of, 214.
,, ,, atrophous, 214.
,, ,, common, 199, 769.
,, ,, compensatory, 220.
,, ,, complementary, 220.
,, ,, hypertrophous, 200.
,, ,, interlobular, 223.
,, ,, interstitial, 223.

Emphysema, forms of, large-lunged, 200.
,, ,, mediastinal, 107.
,, ,, small-lunged, 218.
,, ,, subcutaneous, of neck, 101.
,, ,, vicarious, 220.
,, frequency of, 205.
,, girdle of veins in, 207.
,, hæmoptysis in, 211.
,, hypertrophous, 200.
,, inheritance in, 205.
,, morbid anatomy of, 200.
,, physical signs of, 206.
,, pneumatograms in, 209.
,, pneumonia in relation to, 293.
,, pneumothorax due to, 769.
,, prognosis in, 214.
,, reduction of vital capacity of lungs in, 208.
,, relation of, to morbus cordis, 214.
,, ,, other diseases, 213.
,, ,, phthisis, 213.
,, ,, pneumonia, 213, 293.
,, sex in, 205.
,, symptoms of, 211.
,, theories of, production of, 202.
,, treatment of, 215.
Empyema, 699.
,, absorption, spontaneous, of, 703.
,, aneurysm, abdominal, simulated by, 708.
,, bacteriology of, 713.
,, ,, pneumococcus, 290, 715.
,, ,, staphylococcus, 715.
,, ,, streptococcus, 714.
,, ,, tubercle-bacillus, 716, 749.
,, ,, typhoid-bacillus, 716.
,, characters of effusion in, 702.
,, complications in, 708.
,, ,, abscess of brain, 709.
,, ,, amyloid disease, 711.
,, ,, brain, abscess of, 709.
,, ,, ,, embolism in, 710.
,, ,, diaphragm, inflammation of, 709.
,, ,, empyema of opposite side, 709.
,, ,, hemiplegia, 711.
,, ,, mediastinitis, 708.
,, ,, meningitis, 709.
,, ,, parotid abscess, 709.
,, ,, pericarditis, 708.
,, ,, phrenitis, 709.
,, ,, pneumonia dissecans, 708.
,, ,, septic infection, 709.
,, ,, thrombosis in vessels or heart, 709.
,, course of, 703.
,, diagnosis from abscess of chest wall, 718.
,, ,, aneurysm, 718, 722.
,, ,, hepatic abscess, 718.
,, ,, hydatid of liver, 718.
,, ,, serous effusion, 668.
,, ,, subphrenic abscess, 718.
Empyema, Estländer's operation for, 738.
,, etiology of, 711.
,, forms of, 716.
,, ,, double, 724.
,, ,, fœtid, 726.
,, ,, localised, 717.
,, ,, ,, basic, 717.
,, ,, ,, diaphragmatic, 717.
,, ,, ,, interlobar, 717.
,, ,, ,, pericardium, near, 718.
,, ,, loculated, 716.
,, ,, malignant, 720.
,, ,, metapneumonic, 290.
,, ,, pulsating, 720.
,, ,, recrudescent, 719.
,, ,, recurrent, 719.
,, ,, relapsing, 719.
,, ,, tubercular, 749.
,, mortality, 713.
,, onset, 699.
,, perforation into intestine, 707.
,, ,, ,, liver, 707.
,, ,, ,, mediastinum, 707.
,, ,, ,, œsophagus, 707.
,, ,, ,, pericardium, 707.
,, ,, ,, peritoneum, 707.
,, ,, ,, spleen, 707.
,, ,, ,, stomach, 707.
,, ,, ,, vessels, 707.
,, ,, through chest walls, 706.
,, ,, ,, lung, 704.
,, phthisis in relation to, 526.
,, pneumonia in relation to, 290.
,, pneumothorax in relation to, 768.
,, pointing in, 701.
,, psoas abscess, simulated by, 708.
,, temperature in, 700.
,, tension, intrapleural, in, 636.
,, treatment of, by Estländer's operation, 727, 738.
,, ,, ,, deformity after, 740.
,, ,, ,, results of, 741.
,, ,, incision, 731.
,, ,, ,, anæsthetic for, 728.
,, ,, ,, counter opening in, 731.
,, ,, ,, drainage difficulties after, 736.
,, ,, ,, dressing after, 732.
,, ,, ,, fistula after, 738.
,, ,, ,, management after, 734.
,, ,, ,, operation, the, 729.
,, ,, ,, rashes after, 733.
,, ,, ,, resection of rib in, 729.
,, ,, ,, results of, 737.
,, ,, ,, tube, the, 732.

Empyema, treatment of, by incision, washing out after, 731.
,, ,, paracentesis, 727, 741.
,, ,, perflation, 745.
Enchondroma of lung, 873.
Endocarditis in pneumonia, 291.
,, pneumococcic, 291.
Epidemic pneumonia, 249.
Epithelioma of lung, 878, 880.
Erosion of pulmonary vessels, 386.
Erysipelas, laryngitis in, 37.
Estländer's operation, 738.
,, ,, deformity after, 740.
,, ,, results of, 741.
Excavation, chronic, of lung, 502.
Excision of lung in phthisis, 545.
Expectoration, albuminous, 162, 691.
,, serous, 162, 691.
See also *hæmoptysis*, *sputum*, etc.

Fat embolism, 358.
,, morbid anatomy of, 358.
,, symptoms of, 359.
Femoral vein, thrombosis of, in phthisis, 512.
Fever, treatment of the, in phthisis, 545.
Fevers, the, in relation to bronchitis, 181.
,, ,, pleurisy, 651.
Fibroid degeneration of lung, 317.
,, induration, 317.
,, phthisis, 409, 500.
Fibroma of lung, 872.
Fibrosis of lung, 317.
,, ,, after pneumonia, 260.
,, ,, syphilitic, 838.
Fine hair crepitation in pneumonia, 278.
Fishbone in larynx of infant, 30.
Fistula in ano in phthisis, 511.
Fistula, thoracic, after empyema, 738.
Flat chest, 453.
Flesh, loss of, in phthisis, 459.
Fluxion, collateral, 233, 238.
Fœtal tuberculosis, 451.
Fœtid empyema, 726.
Fœtor in gangrene of lung, 366.
Fogs, effect of, on bronchitis, 113.
Foreign bodies in larynx, 24.
,, ,, course and complications, 28.
,, ,, diagnosis of, 29.
,, ,, impacted for long time, 29.
,, ,, physical signs of, 26.
,, ,, prognosis in, 29.
,, ,, results of, 27.
,, ,, seat of impaction, chief, 25.
,, ,, symptoms of, 25.
,, ,, tracheotomy for, results of, 30.
,, ,, treatment of, 29.
Formic aldehyde in treatment of phthisis, 543.
Frenal ulcer in whooping-cough, 605.
Friction, crepitation a form of, 655.
,, dry leather, 655.
,, in dry pleurisy, 655.
,, pleural, 655.
,, pleuro-pericardial, 655.
,, redux, 667.

Galloping consumption, 494.
Gangrene of the lung, 366.
,, ,, age in, 368.
,, ,, associated with phthisis, 508.
,, ,, bacteriology of, 366.
,, ,, complications in, 373.
,, ,, diagnosis of, 373.
,, ,, etiology of, 368.
,, ,, fœtor in, 366.
,, ,, following pneumonia, 260.
,, ,, lesions associated with, 370.
,, ,, morbid anatomy of, 367.
,, ,, physical signs of, 371.
,, ,, prognosis of, 373.
,, ,, treatment of, 373.
Gelatinous infiltration, 407.
General tuberculosis in phthisis, 510.
Glands discharging into air-tubes, 105.
,, impacted in larynx, 106.
,, pneumothorax, produced by suppurating, 106.
See also *bronchial glands*.
Glottis, œdema of, 53.
Going abroad in phthisis, 555.
Goître, aerial, 99.
,, common, 97.
,, exophthalmic, 99.
,, trachea compressed by, 97.
Gonorrhœa in relation to pleurisy, 652.
Gout in relation to asthma, 575.
,, ,, bronchitis, 183.
,, ,, phthisis, 520.
,, ,, pleurisy, 650.
Granular kidney in phthisis, 520.
Gray granulation of Bayle, 409.
Grinders' phthisis, 198.
Gruyère-cheese lung, 187.
Gumma of lung, 837.

Harrison's furrow, 200.
Hay-asthma, 577.
Hæmatemesis, 377.
Hæmatoma of the pleura, 761.
Hæmoptysis, 375, 466.
,, blood clots in, 376, 464.
,, bronchial, 378.
,, diagnosis of, 376.
,, extrinsic, 377.
,, fatal, 382, 388.
,, forms of, 375, 381.
,, in abscess of lung, 380.
,, ,, acute inflammatory congestion, 380.
,, ,, embolism, 379.
,, ,, emphysema, 211.
,, ,, gangrene of lung, 380.
,, ,, hypostatic congestion, 232.
,, ,, infarct, 379.
,, ,, laryngitis tuberculosa, 81.
,, ,, morbus cordis, 379.
,, ,, plastic bronchitis, 172.
,, ,, whooping-cough, 604.
,, intrinsic, 378.
,, laryngeal, 379.

Hæmoptysis, parasitic, 379.
,, pulmonary, 379.
Hæmoptysis in phthisis, 380, 466, 515.
,, pathology of, 382.
,, profuse, forms of, 389.
,, prognosis of, 391.
,, remittent, 388.
,, suffocative, 388.
,, treatment of, theory of, 396.
,, ,, profuse, 393.
,, ,, slight, 391.
,, ,, with astringents, 394.
,, ,, ,, derivatives, 395.
,, ,, ,, dieting, 396.
,, ,, ,, hæmostatics, 393.
,, ,, ,, sedatives, 394.
,, ,, ,, vascular depressants, 394.
Hæmorrhage in whooping-cough, 604.
,, into mediastinum, 107.
Hæmorrhagic pleural effusion, 759.
,, ,, causes of, 759.
,, ,, diagnosis of, 760.
,, ,, frequency of, 759.
,, ,, prognosis in, 760.
,, ,, treatment of, 760.
Hæmothorax, 761.
,, diagnosis, 763.
,, non-traumatic, 762.
,, prognosis in, 763.
,, results of, 763.
,, signs of, 763.
,, traumatic, 761.
,, treatment of, 764.
Healed tuberculosis, frequency of, 439.
Heart, bronchitis, effect of, on, 128.
,, clots in empyema, 709.
,, ,, pneumonia, 258.
,, disease in relation to phthisis, 516.
,, ,, ,, pneumonia, 294.
,, displacement of, in pleuritic effusion, 663.
,, failure in phthisis, 426, 481.
,, ,, pneumonia, 231.
,, murmurs, caused by dilatation, 130.
,, ,, ,, displacement, 785.
,, venous congestion, effect of, on, 129.
Heart-lung, 235.
Hemiplegia after empyema, 711.
,, in whooping cough, 606.
Hepatic abscess, empyema resembling, 718.
Hernia of lung, 835.
Hiccough, 829.
,, in pneumonia, 274.
Hot-baths in bronchitis, 141.
,, in pneumonia of children, 307.
House pneumonia, 250.
Hydro-pneumothorax, 816.
Hydrops ex vacuo, 674, 757.
,, laryngis, 54.
,, pleuræ, 756.
Hydrops pleuræ adiposus, 765, 767.
,, ,, chylosus, 765.
Hydrothorax, 756.
,, causes of, 756.
,, diagnosis of, 758.
,, prognosis in, 758.
,, symptoms of, 758.
,, treatment of, 759.
,, unilateral, 757.
Hydatid in blood vessels, 864.
,, of liver, empyema resembling, 718.
,, of lung, 860.
,, ,, diagnosis of, 868.
,, ,, duration of, 869.
,, ,, frequency of, 861.
,, ,, hæmoptysis in, 863, 866.
,, ,, physical signs of, 867.
,, ,, rupture of, 862.
,, ,, treatment of, by drugs, 876.
,, ,, ,, incision, 871.
,, ,, ,, paracentesis, 870.
Hyperæmia of lung, 231.
,, ,, collateral, causing bronchitis, 111.
Hyperæsthesia, general cutaneous, in phthisis, 522.
,, ,, in pneumonia, 287.
Hyper-distension of lung, 222.
Hyperpyrexia in pneumonia, 265.
Hypertrophy of lung, compensatory, 220.
,, ,, complementary, 220.
,, ,, displacement of boundaries in, 221.
,, ,, pleurisy leading to, 674.
,, ,, vicarious, 220.
Hypostatic congestion of lung, 231, 240.
,, ,, broncho-pneumonia in, 232.
,, ,, etiology of, 233.
,, ,, hæmoptysis in, 232.
,, ,, infarct in, 232.
,, ,, results of, 232.
Hysterical paralysis of diaphragm, 828.

Incision for empyema, 731.
,, pneumothorax, 818, 821.
,, serous effusion, 696.
Induration, fibroid, of lung, 317.
,, broncho-pneumonia leading to, 329.
,, gray, 320.
,, red, 320.
,, slaty, 321.
Indurative pneumonia, primary, 321.
Infantile laryngeal spasm, 62.
,, stridor, congenital, 62.
Infarct, pulmonary, 351.
,, ,, in hypostatic congestion, 232.
Infiltration, gelatinous, 407.
,, tubercular, 408.
Inflammation of lung, acute, 245.
Influenza in relation to phthisis, 518.
,, ,, pneumonia, 295.
Inhalations in asthma, 596.

Inhalations in bronchitis, 133.
Inheritance in phthisis, 449.
Injections, intratracheal, in bronchitis, 137.
,, ,, phthisis, 539.
Injury in relation to pneumonia, 298, 318.
Insanity and asthma, 581.
Insomnia in pneumonia, 286.
Inspiratory recession in bronchitis, 124.
Interlobar empyema, 717.
Interlobular emphysema, rarity of, 225.
,, pneumonia, acute, 315.
Interstitial emphysema, 223.
,, ,, in whooping-cough, 605.
Interstitial pneumonia, acute, 314.
,, ,, ,, after bronchitis, 317.
,, ,, ,, broncho-pneumonia, 317.
,, ,, ,, compression of lung, 317.
,, ,, ,, injury, 318.
,, ,, ,, pneumonia, acute lobar, 260, 318.
,, ,, ,, pneumonokoniosis, 317.
,, ,, ,, syphilis, 318.
,, ,, ,, tuberculosis, 318.
Interstitial pneumonia, chronic, 318.
,, ,, ,, diagnosis of, 319.
,, ,, ,, pathological change in, 318.
,, ,, ,, physical signs of, 319.
,, ,, ,, prognosis in, 319.
,, ,, ,, symptoms of, 319.
Intestine, empyema bursting into, 707.
,, perforation of, in phthisis, 510.
,, tubercular ulceration of, in phthisis, 421.
Intra-tracheal injections in bronchitis, 137.
,, ,, phthisis, 539.

Jaundice in pneumonia, 284.
Junod's boot, 395.

Kidney disease in relation to phthisis, 294.
,, ,, ,, pneumonia, 294.
Knife-grinders' phthisis, 198.
,, ,, duration of life in, 198.
,, ,, mortality in, 198.

Lactation in relation to phthisis, 519.
Laryngeal diphtheria, 39.
,, phthisis, 74.
,, ,, primary, 79.
Laryngismus stridulus, 58.
,, ,, diagnosis of, 59.
,, ,, etiology of, 59.
,, ,, pathology of, 58.
,, ,, prognosis of, 59.
,, ,, theories of, 62.
,, ,, treatment of, 60.
Laryngitis, 31.
,, in the adult, acute, 32.
,, ,, chronic, 33.
,, ,, treatment, 31, 34.
,, in the child, 35.
,, ,, treatment of, 36.
,, in phthisis, 509.
,, in specific fevers, 37.
,, membranous (see also *membranous laryngitis*), 38.
,, œdematous, 55.
,, sicca, 33.
,, singers' nodes in, 34.
,, spasmodic, 35.
,, subglottic, 34.
,, tubercular, 74, 419.
,, ,, age in, 79.
,, ,, diagnosis of, 81.
,, ,, frequency in phthisis, 78.
,, ,, hæmoptysis in, 81.
,, ,, pathology of, 75.
,, ,, polypoid vegetation in, 77.
,, ,, prognosis in, 81.
,, ,, sex influence of, 79.
,, ,, symptoms of, 80.
,, ,, treatment of, 82.
Laryngorrhœa, 33.
Larynx, carcinoma of, 93.
,, ,, erasion for, 95.
,, ,, excision for, 95.
,, cicatricial stricture of, 88.
,, cysts of, 92.
,, fibromata of, 91.
,, fishbone in, in infant, 30.
,, gland impacted in, 106.
,, nervous affections of, 56.
,, œdema of, 53.
,, papilloma of, 91.
,, sarcoma of, 93.
,, stricture of, 88.
,, syphilis of, in adult, 84.
,, ,, in child, 85.
,, tumours of, malignant, 91.
,, ,, non-malignant, 93.
Latent phthisis, 504.
,, pneumothorax, 807.
Lead-poisoning in relation to phthisis, 520.
Left auricle, dilated, pressing on root of lung, 108.
Leptothrix pulmonalis, 163.
Leyden's crystals in asthma, 569.
Lip, tubercular ulceration of, in phthisis, 424.
Lipoma of the lung, 873.
Liver, cirrhosis of, in phthisis, 426.
,, empyema perforating into, 707.
Lobar pneumonia, chronic, 320.
Locality, effect of, in asthma, 597.
Luftkropf, 99.
Lung, abscess of, 362.
,, actinomycosis of, 841.
,, anatomy of, 1.
,, aspergillosis of, 856.
,, bilharzia hæmatobia of, 860.
,, blood vessels of, 6–9.

ung, carcinoma of, 882.
,, carnified, 644.
,, cavities in, 410.
,, cirrhosis of, 317, 409.
,, coccidiosis of, 859.
,, elasticity of, 775.
,, enchondroma of, 873.
,, excavation, chronic, of, 502.
,, fibroid degeneration of, 317.
,, ,, induration of, 317.
,, fibroma of, 872.
,, fibrosis of, 317.
,, gangrene of, 366.
,, gumma of, 837.
,, heart, the, 235.
,, hernia of, 835.
,, hydatids of, 860.
,, hyper-distension of, 232.
,, lipoma of, 873.
,, lobes of, in relation to chest walls, 3.
,, lymphatics of, 7.
,, mucor corymbifer in, 859.
,, mycosis of, 840.
,, nerves of, 10.
,, new-growths of, 872.
,, oïdium albicans in, 859.
,, osteoma of, 870
,, paralysis of, 223.
,, perforation of, by empyema, 704.
,, relaxed, 222.
,, root of, 10.
,, rupture of, experimental, 223.
,, ,, traumatic, not always causing pneumothorax, 774.
,, sarcoma of, 874.
,, streptococcus of, 854.
,, syphilis of, 836.
,, weight of, 2.
,, ,, in pneumonia, 256.
Lupus in relation to phthisis, 520.
Lymphangitis pulmonalis, 315.
Lymphatics of lungs, 7.
Lysis in pneumonia, 268.

Madeira for the phthisical, 560.
Malarial pneumonia, 296.
Mania in pneumonia, 292.
Measles, laryngitis in, 37.
,, relation of, to phthisis, 518.
,, ,, pneumonia, 295.
,, ,, whooping-cough, 609.
Median position of vocal cords, conditions causing, 65.
Mediastinum, abscess of, 106.
,, affections of, in association with empyema, 707.
,, emphysema of, 107.
,, hæmorrhage in, 107.
,, tumour of, pressing on trachea, 102.
Melancholia in pneumonia, 292.
Membranous laryngitis (diphtheritic), 39.
,, age in, 40.
,, chronic, 47.
,, complications of, 45.
,, course of, 44.
Membranous laryngitis, death, causes of, in, 45
,, duration of, 44.
,, mortality of, 51.
,, pathology of, 41.
,, pneumonia in relation to, 295.
,, prognosis of, 47.
,, quarantine in, 52.
,, sequelæ of, 46.
,, sex in, 40.
,, symptoms of, 43.
,, treatment of, 49.
,, ,, antitoxin, 50.
,, ,, during convalescence, 51.
,, ,, effect on mortality of, 51.
,, ,, intubation, 49.
,, ,, tracheotomy, 49.
,, primary tracheal, 47.
Meningitis after empyema, 709.
,, ,, pneumonia, 292.
,, tubercular in phthisis, 510.
Menstruation in phthisis, 519.
,, vicarious, and hæmoptysis, 380.
Mercury in treatment of phthisis, 542.
Metallic tinkling in pneumothorax, 789.
Metapneumonic empyema, 290.
Metastatic pneumonia, 312, 323.
Miliary tuberculosis, acute, 495, 510.
Mitral disease, congestion of lungs in, 235.
Morbus cordis, bronchitis due to, 179.
,, ,, in relation to pneumonia, 293.
Mortality in bronchitis, 116.
,, empyema, 712.
,, gangrene of lung, 116, 373.
,, membranous laryngitis, 51.
,, phthisis, 442.
,, ,, diminution in, 446.
,, pleurisy, 647.
,, pneumonia, 303.
,, pneumothorax, 795.
,, whooping-cough, 611.
Motions, grass green, in pneumonia, 284.
Mucor corymbifer in lung, 859.
Murmurs, cardiac, due to dilatation, 130.
,, ,, ,, displacement of heart, 663.
,, ,, tricuspid, 130.
,, venous, produced by pressure of enlarged bronchial glands, 105.
Mycosis of lung, 840.
Myoidema, 840.

Narcotics in bronchitis, 142.
Neck, emphysema of, 101.
Necrosis of cartilages of air-tubes, 70.
Nephritis, acute, in pneumonia, 292.
Nerves of lung, 10.
Nervous diseases in relation to phthisis, 522.
,, ,, ,, to pneumonia, 298.
,, ,, of larynx, 56.

Neuritis, peripheral, in phthisis, 522.
Neuroses, hysterical, 516.
New-growth of bronchial glands, 104.
New-growths of lung and pleura, 872.
,, ,, complications of, in lung, 891.
,, ,, ,, in pleura, 891.
,, ,, diagnosis of, 890.
,, ,, duration of, 891.
,, ,, fatality of, 892.
,, ,, forms of, 872.
,, ,, ,, cancer, 877.
,, ,, ,, enchondroma, 873.
,, ,, ,, fibroma, 872.
,, ,, ,, lipoma, 873.
,, ,, ,, osteoma, 873.
,, ,, ,, sarcoma, 874, 875.
,, ,, in mediastinum, 102.
,, ,, physical signs of, 889.
,, ,, symptoms of, 887.
,, ,, treatment of, 892
New Zealand for the phthisical, 561.
Night sweats in phthisis, 460.
Nitre in asthma, 595.
Nitrite of amyl, 594.
Nitro-glycerine, 594.
Nummular sputum in phthisis, 463.

Obstruction of air-tubes, 20, 97.
Obstruction, respiratory, in nose, 19.
,, ,, pharynx, 19.
Œdema of the glottis, 53.
,, ,, lung, acute, causes of, 242.
,, ,, ,, chronic, 242.
,, ,, ,, hypostatic, 231, 240.
,, ,, ,, solid, 240.
Œdematous laryngitis, 55.
,, ,, diagnosis of, 56.
,, ,, pathology, 55.
,, ,, symptoms, 56.
,, ,, treatment, 56.
Œsophageal pouches, 100.
Œsophagus, foreign bodies in, 101.
,, new-growth of, pressing on trachea, 101.
,, perforated by empyema, 707.
,, tubercular ulceration of, in phthisis, 423.
Oïdium albicans in lung, 859.
One-day pneumonia, 301.
Open-air treatment of phthisis, 564.
Opium in asthma, 592.
,, whooping-cough, 615.
Osteo-arthropathy, pulmonary, 493.
Osteoma of lung, 873.
Osteo-sarcoma of lung, 875.
Otitis media in pneumonia, 292.
Overcrowding in relation to phthisis, 441.

Pachydermia laryngis, 34.
Palate, tubercular ulceration of, in phthisis, 425.
Paracentesis, dry, 669.
,, empyema treated by, 741.
Paracentesis, pneumothorax treated by, 818.
Paralysis abductor, 65.
,, hysterical, 828.
,, of diaphragm, 830.
,, of lung, 223, 239.
Paralytic thorax, 453.
Parotid abscess after empyema, 709.
,, ,, pneumonia, 292.
Pearly sputum, 160.
Perflation, empyema treated by, 745.
Perforation of intestine in phthisis, 510.
Peribronchitis fibrosa, 318.
Pericardial effusion pressing on root of lung, 107.
Pericarditis in empyema, 707.
,, pneumonia, 291.
Perichondritis of larynx, 72.
,, ,, diagnosis of, 73.
,, ,, prognosis in, 73.
,, ,, symptoms of, 72.
,, ,, treatment of, 73.
,, of trachea and bronchi, 74.
Periodic respiration, 617.
Peripheral neuritis in phthisis, 522.
Peripneumonia, 312.
,, notha, 155.
Peritoneum, empyema bursting into, 707.
Peritonitis, tubercular, in phthisis, 510.
Pertussis, 600.
Phlegmonous inflammation in neck, 101.
Phlegmorrhagia pulmonum, 160.
Phthisis, 494.
,, acute, prognosis of, 533.
,, Addison's disease in, 522.
,, age in relation to, 455.
,, albuminuria in, 486.
,, alcoholism in, 521.
,, amyloid disease in, 511.
,, antiseptics in, 542.
,, aphonia in, 508.
,, appetite in, 483.
,, apyrexial, 458.
,, arsenic in, 543.
,, attendants, infection of, in, 437.
,, basic, 414.
,, bedsores in, 512.
,, blood in, 487.
,, blood-casts in, 464.
,, bowels in, 484.
,, Bright's disease in, 520.
,, bronchial atresia in, 418.
,, ,, casts in, 464.
,, ,, glands in, 419.
,, bronchiectasis in, 418.
,, bronchitis in, 514.
,, broncho-pneumonia in, 514.
,, broncho-pneumonic, 499.
,, bucco-pharyngeal, tuberculosis in, 421.
,, calcareous masses in sputum of, 465.
,, cancer, relation of, to, 517.
,, cattle, infection of man from, 431.
,, children's, 454.
,, chronic, 499.
,, ,, prognosis in, 532.
,, climate in, 440, 552.

Phthisis, clubbing in, 488.
,, complementary emphysema in, 418.
,, complications of, 505.
,, ,, acute pulmonary tuberculosis, 510.
,, ,, amyloid disease, 511.
,, ,, aphonia, 508.
,, ,, bedsores, 512.
,, ,, fistula, 511.
,, ,, gangrene of lung, 508.
,, ,, general tuberculosis, 510.
,, ,, laryngitis, 509.
,, ,, miscellaneous affections, 512.
,, ,, perforation of intestines, 510.
,, ,, pleuritic effusion, 507.
,, ,, pneumothorax, 506.
,, ,, ,, latent, 507.
,, ,, thrombosis of femoral vein, 512.
,, ,, tubercular meningitis, 510.
,, ,, ,, peritonitis, 510.
,, ,, ,, ulceration of bowel, 510.
,, cough in, 461.
,, ,, treatment of, 547.
,, course of, 523.
,, creasote in, 543.
,, cyanosis in, 480.
,, death, modes of, in, 525.
,, diabetes mellitus and, 523.
,, diagnosis of, 527.
,, diarrhœa in, 485.
,, ,, treatment of, 550.
,, diathesis in, 452.
,, duodenum, tubercular ulceration of, in, 423.
,, duration of, 523.
,, dyspeptic, 483.
,, dysphagia in, 484.
,, dyspnœa in, 480.
,, empyema in, 213.
,, etiology of, 427.
,, fever in, 456.
,, ,, treatment of, 545.
,, fevers, specific, in relation to, 517.
,, fibroid, 409, 500.
,, fistula in, 511.
,, florida, 499.
,, formic aldehyde, treatment of, by, 543.
,, forms of, 494.
,, ,, acute, 533.
,, ,, chronic, 748.
,, ,, galloping, 494.
,, ,, subacute, 748.
,, galloping, 494.
,, gangrene of lung in, 418, 508.
,, general condition in relation to, 427, 444.
,, generative system in, 487.
,, gout in relation to, 520.
,, grinders', 198.
,, guaiacol, treatment of, by, 543.
,, hæmoptöe (ab), 515.
Phthisis, hæmoptysis in, 466.
,, ,, relation to, as causing, 515.
,, hair in, 488.
,, hand in, 488.
,, health, feeble, in relation to, 517.
,, heart in, 426.
,, ,, disease of, in relation to, 516.
,, history of, 399.
,, hypertrophy of lung, complementary, in, 418, 477.
,, infection from cattle, 431.
,, ,, from man, 435.
,, ,, from man to man, 436.
,, influenza, relation of, to, 518.
,, inheritance in, 449.
,, injection, intra-laryngeal treatment of, by, 544.
,, ,, intra-pulmonary treatment of, by, 544.
,, institutions, phthisis in, 437.
,, kidney disease, relation of, to, 521.
,, lactation, relation of, to, 519.
,, laryngitis in, 509.
,, latent, 504.
,, lead poisoning in relation to, 520.
,, lip, tuberculosis of, in, 424.
,, liver, amyloid, in, 426.
,, ,, cirrhosis of, in relation to, 426.
,, ,, fatty, in, 425.
,, ,, tubercular, in, 426.
,, loss of flesh in, 459.
,, lupus in relation to, 520.
,, measles, relation of, to, 518.
,, meningitis, tubercular, in, 510.
,, menstruation in, 519.
,, mercury in treatment of, 542.
,, millstone-makers', 198.
,, miners', 197.
,, mortality of, 442, 444, 454.
,, ,, diminution of, 446.
,, murmurs in, cardiac, 483.
,, ,, subclavian, 482.
,, myoidema in, 491.
,, nerve diseases in relation to, 521.
,, nervous system in, 486.
,, night sweats in, 460.
,, ,, ,, treatment of, 541.
,, occupation, relation of, to, 444.
,, œsophagus, tubercular ulceration of, in, 423.
,, open-air treatment of, 554.
,, over-crowding in relation to, 441.
,, pain in chest in, 480.
,, ,, on coughing in, 462.
,, palate, tubercular ulceration of, in, 424.
,, paroxysmal coughing in, 462.
,, pathology of, 401.
,, ,, associated, of, 417.
,, perforation of intestines in, 510.
,, peritonitis, tubercular, in, 510.
,, physical signs of, 469, 479.
,, pleura, tubercle of, in, 420.
,, pleurisy, relation of, to, 514.
,, pleuritic effusion in, 420, 507.

Phthisis, pneumonia, acute, in, 513.
,, pneumonic, acute, 499.
,, pneumothorax in, 421, 506.
,, ,, latent, in, 507.
,, population, density of, in relation to, 441.
,, potters', 198.
,, pregnancy in relation to, 519.
,, preventive, treatment of, 540.
,, prognosis of, in general, 531.
,, ,, acute, 533.
,, ,, chronic, 532.
,, prophylaxis of, 536.
,, pulse in, 481.
,, relation of, to Addison's disease, 522.
,, ,, age, 453.
,, ,, alcoholism, 521.
,, ,, Bright's disease, 520.
,, ,, bronchitis, 514.
,, ,, broncho - pneumonia, 514.
,, ,, cancer, 517.
,, ,, climate, 440.
,, ,, density of population, 441.
,, ,, diabetes mellitus, 523.
,, ,, diathesis, 452.
,, ,, emphysema, 213.
,, ,, empyema, 526.
,, ,, feeble health, 517.
,, ,, fevérs, specific, 517.
,, ,, general condition of health, 444.
,, ,, gout, 520.
,, ,, hæmoptysis, 515.
,, ,, heart disease, 516.
,, ,, influenza, 518.
,, ,, inheritance, 449.
,, ,, lactation, 519.
,, ,, lead-poisoning, 520.
,, ,, lupus, 520.
,, ,, measles, 518.
,, ,, menstruation, 519.
,, ,, nervous diseases, 521.
,, ,, occupation, 444.
,, ,, pleurisy, 514.
,, ,, pneumonia, 293, 513.
,, ,, pregnancy, 519.
,, ,, race, 439.
,, ,, renal disease, 520.
,, ,, rheumatic fever, 520.
,, ,, sex, 455.
,, ,, syphilis, 516.
,, ,, typhoid fever, 518.
,, ,, unsanitary conditions, 445.
,, rigors in, 461.
,, sanatoria treatment of, 554.
,, sex in relation to, 455.
,, shivering in, 458.
,, signs, general, of, 456.
,, ,, physical, of, 469.
,, skin in, 487.
,, spleen in, 426.
,, spread, mode of, in lung, 415.
,, sputum in, 462.
Phthisis, sputum in, constituents of, 463.
,, stomach, tubercular ulceration of, in, 423.
,, strength, loss of, in, 460.
,, subclavian murmur in, 482.
,, suppuration of lung in, 418.
,, surgical operations for, 545.
,, sweating, night or sleep, in, 460.
,, ,, ,, treatment of, 546.
,, syphilis in relation to, 516.
,, syphilitic, 516, 839.
,, thrombosis of veins in, 427, 512.
,, tongue, tubercular ulceration of, in, 421.
,, treatment of, 536.
,, ,, antiseptic, 542, 544.
,, ,, climatic, 551, 554.
,, ,, open-air, 554.
,, ,, preventive, 540.
,, ,, surgical, 545.
,, ,, therapeutic, 540.
,, ,, by arsenic, 543.
,, ,, creasote, 543.
,, ,, formic aldehyde, 543.
,, ,, guaiacol, 543.
,, ,, mercury, 542.
,, ,, tuberculin, 540.
,, treatment of cough in, 547.
,, ,, diarrhœa in, 550.
,, ,, fever in, 545.
,, ,, sweating in, 546.
,, ,, vomiting in, 549.
,, tubercular ulceration of air-tubes in, 418.
,, ,, ,, bucco-pharynx in, 424.
,, ,, ,, duodenum in, 423.
,, ,, ,, intestine in, 421.
,, ,, ,, œsophagus in, 423.
,, ,, ,, stomach in, 421.
,, tuberculin treatment of, 540.
,, tuberculosis, acute pulmonary, 510.
,, ,, general, in, 510.
,, typhoid fever, diagnosis of, from, 518.
,, ulceration, tubercular, 510.
,, unsanitary surroundings in relation to, 445.
,, urine in, 485.
,, vaccination in relation to, 517.
,, valve-shock in, 482.
,, veins, thrombosis, of, 427, 512.
,, vomiting, 483.
Pigeon-breast, result of atelectasis, 230.
Pilocarpine in asthma, 593.
Pituitary catarrh, 160.
Plastic bronchitis, 167.
,, acute form of, 174.
,, associated lesions of, 169.
,, asthma resembling, 178.
,, cases of, 177.
,, casts in, 168.
,, chronic, 175.
,, diagnosis of, 176.
,, duration of, 175.
,, etiology of, 170.

Plastic bronchitis, hæmoptysis in, 172.
,, pathology of, 169.
,, prognosis of, 175.
,, results of, 170.
,, signs of, 168.
,, symptoms of, 171.
,, temperature in, 173.
,, treatment of, 178.
Pleura, 625.
,, actinomycosis of, 841, 852.
,, anatomy of, 9.
,, cancer of, 885.
,, classification of affections of, 637.
,, cohesion between layers of, 775.
,, effusion into, in phthisis, 420.
,, hæmatoma of, 761.
,, new-growth of, 872.
,, syphilis of, 836.
Pleurisy, acute inflammatory, 640.
,, ,, effect on adjacent organs, 643.
,, ,, etiology of, 646.
,, ,, injury causing, 650.
,, ,, latent, 656.
,, ,, mortality of, 646.
,, ,, pathology of, 641.
,, ,, relation to Bright's disease, 650.
,, ,, ,, gonorrhœa, 651.
,, ,, ,, gout, 650.
,, ,, ,, rheumatic fever, 651.
,, ,, ,, specific fevers, 651.
,, ,, ,, syphilis, 651.
,, ,, ,, tubercle, 652.
,, ,, results, pathological, of, 645.
,, diaphragmatic, 750.
,, double, 753.
,, dry, 640.
,, ,, diagnosis of, 657.
,, ,, etiology of, 646.
,, ,, onset of, 656.
,, ,, pathology of, 640.
,, ,, physical signs of, 654.
,, ,, seat of, 657.
,, ,, symptoms of, 654.
,, ,, temperature in, 656.
,, ,, treatment of, 658.
,, hæmorrhagic, 759.
,, polymorphic, 514.
,, relation to phthisis, 514.
,, ,, pneumonia, 289, 293.
,, tubercular, 746.
,, ,, bacteriology of, 747.
,, ,, clinical types of, 748.
,, ,, ,, acute, 748.
,, ,, ,, chronic, 748.
,, ,, ,, subacute, 748.
,, ,, ,, with empyema, 749.
,, ,, ,, ,, serous effusion, 748.
,, ,, ,, ,, thickening, 749.
,, ,, diagnosis of, 747.
,, ,, pathology of, 746.
,, ,, treatment of, 750.
Pleuritic effusion, serous, 668.
,, ,, bronchial breathing in, 665.
Pleuritic effusion, bruit de pot fêlé in, 665.
,, ,, cardiac murmurs, from displacement in, 663.
,, ,, clotting in heart in, 678.
,, ,, complications of, 678.
,, ,, consequences on affected lung of, 665.
,, ,, ,, opposite lung of, 666.
,, ,, contraction of side after, 672.
,, ,, death in, cause of, 675.
,, ,, ,, mode of, 675.
,, ,, ,, sudden, 675.
,, ,, diagnosis between, and empyema, 668, 679.
,, ,, ,, ,, and pneumonia, 305.
,, ,, displacement of heart in, 663.
,, ,, ,, ,, organs after, 672.
,, ,, ,, ,, during, 661.
,, ,, duration of, 674.
,, ,, effect of, on adjacent organs, 463.
,, ,, etiology of, 646.
,, ,, intra-pleural tension in, 633.
,, ,, kinking of vena cava in, 663.
,, ,, mortality of, 675.
,, ,, pathology of, 642.
,, ,, prognosis of, 678.
,, ,, rate of effusion in, 670.
,, ,, signs, physical, of, in general, 659.
,, ,, ,, ,, large, 660.
,, ,, ,, ,, moderate, 659.
,, ,, skodaic resonance in, 665.
,, ,, temperature in, 671.
,, ,, treatment of, 681.
,, ,, ,, by free incision, 696.
,, ,, ,, paracentesis, 683.
,, ,, ,, ,, contra-indications to, 685.
,, ,, ,, ,, death, sudden, in, 694.
,, ,, ,, ,, frequency of, in general, 685.
,, ,, ,, ,, indications for, 684.
,, ,, ,, ,, ,, for stopping, 689.
,, ,, ,, ,, mode of operation, 688.
,, ,, ,, ,, operation, 687.
,, ,, ,, ,, physical signs after, 694.
,, ,, ,, ,, place of puncture for, 688.
,, ,, ,, ,, pneumothorax after, 695.
,, ,, ,, ,, results of, 698.
,, ,, ,, ,, risks of, 689.
,, ,, ,, ,, rupture of lung during, 687.
,, ,, ,, ,, syphonage, 687.
,, ,, ,, ,, treatment after, 698.
,, ,, with phthisis, 507.
,, ,, with pneumothorax, often serous, 793.
Pleuritis sicca, *cf.* pleurisy, dry, 640.

Pleuro-pneumonia, 245.
Pneumatic treatment of emphysema, 216.
Pneumatocele, 99.
Pneumatograms in emphysema, 209.
Pneumonia—acute, croupous, lobar, pleuro-, 245.
,, ,, abscess of lung after, 259.
,, ,, age, effect of, on prognosis in, 301.
,, ,, aged, in, 300.
,, ,, albuminuria in, 285.
,, ,, alcohol in treatment of, 308, 309.
,, ,, alcoholics in, 297.
,, ,, antipyretics in treatment of, 307.
,, ,, antitoxin treatment of, 306.
,, ,, apex, 299.
,, ,, ,, greater frequency of, in children, 258.
,, ,, aphasia in course of, 287.
,, ,, apyretic, 265.
,, ,, arterial tension in, 280.
,, ,, bacteriology of, 248.
,, ,, bathing, cold, in treatment of, 307.
,, ,, bleeding in, in treatment of, 308.
,, ,, blood, changes in, in, 282.
,, ,, bronchitis in, 279, 290, 293.
,, ,, ,, diagnosis from, 305.
,, ,, broncho-pneumonia, diagnosis from, 305.
,, ,, bruit de pot fêlé in, 277.
,, ,, caffeine in treatment of, 310.
,, ,, cardiac clotting in, 258, 292.
,, ,, ,, failure in, 281.
,, ,, ,, ,, treatment of, 303.
,, ,, Cheyne-Stokes breathing, 287.
,, ,, children in, 300.
,, ,, chlorides in urine in, 285.
,, ,, cold baths in, 307.
,, ,, complications of, 259, 288.
,, ,, congestion, general, of lungs, preceding, 279.
,, ,, ,, hypostatic, in, 279.
,, ,, cough in, 273.
,, ,, course of, 260.
,, ,, cradling in, 307.
,, ,, crisis in, 265.
,, ,, ,, critical discharges in, 269.
,, ,, ,, duration of, 268.
,, ,, ,, hæmorrhage at time of, 267.
,, ,, ,, lysis, relative frequency of, and, 268.
,, ,, ,, œdema of lung at time of, 267.
,, ,, ,, one hour, 268.
,, ,, ,, pneumococcus, relation of, to, 270.
,, ,, critical days in, 269.
,, ,, ,, discharges in, 265.
,, ,, death in, 302.
,, ,, ,, risks of, during convalescence, 303.
,, ,, decubitus in, 273.
,, ,, delirium in, 286, 302.
,, ,, ,, treatment of, 311.
Pneumonia—acute (*continued*).
,, ,, delirium tremens in, 305.
,, ,, diagnosis of, 304.
,, ,, diarrhœa in, 283.
,, ,, diet in, 306.
,, ,, digestive system in, 283.
,, ,, digitalis in, 310.
,, ,, diphtheria, relation of, to, 295.
,, ,, drinkers, of, 300.
,, ,, embolism in, 298.
,, ,, emphysema in relation to, 213, 293.
,, ,, endocarditis in, 291.
,, ,, epidemic, 249.
,, ,, epidemic meningitis in relation to, 293.
,, ,, fevers, specific, diagnosis from, 305.
,, ,, ,, relation to, of, 247, 295.
,, ,, fibrosis following, 260.
,, ,, forms of, 298.
,, ,, ,, aged, in, 300.
,, ,, ,, apex, 299.
,, ,, ,, children, in, 300.
,, ,, ,, drinkers, in, 300.
,, ,, ,, relapsing, 299.
,, ,, ,, wandering, 299.
,, ,, frequency of, 245.
,, ,, gangrene of lung after, 260.
,, ,, granular kidney and, 285.
,, ,, green grass motions in, 283.
,, ,, headache in, 286.
,, ,, heart, in, 258.
,, ,, ,, dilatation of, 281.
,, ,, ,, disease, in relation to, 294.
,, ,, ,, failure in, 281.
,, ,, hepatisation, gray and red, 254.
,, ,, herpes in, appearing after crisis, 288.
,, ,, ,, date of, 288.
,, ,, ,, position of, 288.
,, ,, hiccough in, 274.
,, ,, history of, 311.
,, ,, house, 250.
,, ,, hyperæsthesia, cutaneous, in, 289.
,, ,, hyperpyrexia in, 265.
,, ,, ice in treatment of, 310.
,, ,, incubation period of, 253.
,, ,, infarct in, 298.
,, ,, infection, direct, in, 251.
,, ,, influenza in relation to, 295.
,, ,, injury in relation to, 298.
,, ,, insomnia in, 286.
,, ,, ,, treatment of, 311.
,, ,, interstitial changes, following, 260.
,, ,, jaundice in, 283.
,, ,, kidney disease in relation to, 294.
,, ,, ,, granular, in, 285.
,, ,, lysis, termination of, by, 268.
,, ,, malaria in relation to, 296.
,, ,, mania in, 293.
,, ,, measles in relation to, 295.
,, ,, melancholia in, 293.
,, ,, meningitis in, 292.

Pneumonia—acute (*continued*).
,, ,, meningitis in, cerebro-spinal, in, 293.
,, ,, mortality of, 303.
,, ,, ,, in relation to age, 304.
,, ,, ,, ,, complications, 304.
,, ,, ,, ,, extent, 304.
,, ,, ,, ,, seat, 304.
,, ,, ,, ,, sex, 304.
,, ,, motions, grass green, in, 280.
,, ,, narcotics in treatment of, 311.
,, ,, nephritis, acute, in, 292.
,, ,, nervous diseases in, 298.
,, ,, œdema of lung at time of crisis of, 267.
,, ,, one-day, 301.
,, ,, onset of, 263.
,, ,, ,, favourite time of, 264.
,, ,, ,, sudden death at time of, 264.
,, ,, otitis media in, 292.
,, ,, oxygen in treatment of, 309.
,, ,, packing, cold, in treatment of, 307.
,, ,, pain in, 270.
,, ,, ,, treatment of, 307, 310.
,, ,, parotid abscess in, 292.
,, ,, parts of lung attacked in, 287.
,, ,, pathology of, 253.
,, ,, pericarditis in, 291.
,, ,, phthisis in relation to, 293, 305.
,, ,, plague, 312.
,, ,, pleurisy in, 289.
,, ,, ,, relation of, to, 295.
,, ,, pleuritic effusion in, 289, 305.
,, ,, pneumothorax in, 291.
,, ,, precritical drop in temperature of, 264.
,, ,, pregnancy in relation to, 298.
,, ,, preventive treatment of, 306.
,, ,, prognosis of, 301.
,, ,, ,, in relation to age, 301.
,, ,, ,, ,, delirium, 302.
,, ,, ,, ,, rate of breathing, 302.
,, ,, ,, ,, rate of pulse, 302.
,, ,, ,, ,, sputum, changes in, 302.
,, ,, ,, ,, temperature, 302.
,, ,, ,, ,, tremors, 302.
,, ,, ,, ,, typhoid state, the, 302.
,, ,, pseudocrisis, 264.
,, ,, pulse rate in, 283, 312.
,, ,, pulse-respiration ratio in, 271.
,, ,, pyæmia in, 293.
,, ,, relapses in, 299.
,, ,, relapsing form of, 299.
,, ,, relation to other diseases, 293.
,, ,, ,, alcoholism, 297.
,, ,, ,, bronchitis, 293.
,, ,, ,, diphtheria, 295.

Pneumonia—acute (*continued*).
,, ,, relation to emphysema, 213, 293.
,, ,, ,, fevers, the specific, 295.
,, ,, ,, heart disease, 294.
,, ,, ,, influenza, 295.
,, ,, ,, injury, 298.
,, ,, ,, kidney disease, 294.
,, ,, ,, malaria, 296.
,, ,, ,, measles, 295.
,, ,, ,, nervous diseases, 298.
,, ,, ,, phthisis, 293, 513.
,, ,, ,, pleurisy, 293.
,, ,, ,, pregnancy, 298.
,, ,, ,, rheumatic fever, 294.
,, ,, ,, scarlet fever, 295.
,, ,, ,, typhoid fever, 295.
,, ,, resolution in, 259.
,, ,, respiration-rate in, 271.
,, ,, ,, prognosis in relation to, 302.
,, ,, rheumatic fever and, 264.
,, ,, rigor in, 263.
,, ,, scarlet fever in relation to, 295.
,, ,, season in relation to, 245.
,, ,, sex in relation to, 245.
,, ,, shivering in, 263.
,, ,, signs, physical, of, 263.
,, ,, skin in, 287.
,, ,, ,, gangrene of, 288.
,, ,, ,, eruptions in, 288.
,, ,, ,, ,, disappearance of, at crisis, 288.
,, ,, ,, ,, erythema, 288.
,, ,, ,, ,, herpes, 288.
,, ,, ,, ,, pemphigus, 288.
,, ,, skodaic resonance in, 277.
,, ,, sponging in, 307.
,, ,, sputum in, 274.
,, ,, ,, amount of, 275.
,, ,, ,, bile-stained, 275.
,, ,, ,, hæmorrhagic, 275.
,, ,, ,, in relation to prognosis, 302.
,, ,, ,, prune-juice, 275.
,, ,, ,, rusty, 275.
,, ,, stimulants in treatment of, 301, 309.
,, ,, strophanthus in treatment of, 310.
,, ,, subsultus tendinum in, 287.
,, ,, sweating in, 288.
,, ,, symptomatic treatment of, 306.
,, ,, symptoms of, 270.
,, ,, temperature in, 261.
,, ,, ,, charts, 266.
,, ,, ,, crisis, 265.
,, ,, ,, ,, taken every hour at, 268.
,, ,, ,, fall, 265.
,, ,, ,, precritical drop, 264.
,, ,, ,, prognosis in relation to, 302.
,, ,, ,, pseudocrisis, 264.
,, ,, ,, remittent, 264.
,, ,, ,, rise, 264.

Pneumonia—acute (*continued*).
,, ,, thrombosis of heart in, 258.
,, ,, treatment of, 306.
,, ,, ,, alcohol in, 308, 309.
,, ,, ,, antipyretics in, 307.
,, ,, ,, antitoxine in, 306.
,, ,, ,, baths in, 307.
,, ,, ,, bleeding in, 308.
,, ,, ,, caffeine in, 310.
,, ,, ,, cold baths, sponging, etc., in, 307.
,, ,, ,, cradling in, 307.
,, ,, ,, diet in, 306.
,, ,, ,, digitalis in, 310.
,, ,, ,, ice in, 310.
,, ,, ,, narcotics in, 311.
,, ,, ,, oxygen in, 309.
,, ,, ,, preventive, 306.
,, ,, ,, stimulants in, 306, 309.
,, ,, ,, strophanthus in, 310.
,, ,, ,, symptomatic, 306.
,, ,, tremors in, 302.
,, ,, tympanites in, 287.
,, ,, typhoid fever in relation to, 295.
,, ,, typhoid state, the, in, 302.
,, ,, urea in, 284.
,, ,, urine in, 284.
,, ,, vomiting in, 283.
,, ,, wandering, 299.
,, ,, weights of lungs in, 256.
Pneumonia alba, 836.
,, caseating, 407.
,, chronic, 320.
,, ,, alveolar, 320.
,, ,, interstitial, 320.
,, ,, lobar, 320.
,, ,, forms of, 320.
,, desquamative, 323, 407.
,, dissecans, empyema leading to, 708.
,, lobar, 245.
,, metastatic, 323.
,, ,, abscesses in, 313.
,, pleura, 245.
,, primary indurative, 321
,, serosa, 234.
Pneumococcus, the, 248, 270.
,, relation to crisis, in pneumonia, 270.
,, empyema, 290, 715.
Pneumo-paresis, 233, 239.
Pneumo-koniosis, 192.
,, analysis, chemical, of lungs, 198.
,, forms of, 197.
,, history of, 192.
,, pathology of, 193.
,, signs, physical, of, 196.
,, symptoms of, 195.
,, treatment of, 199.
Pneumono-mycoses, 840.
Pneumothorax, 767.
,, abscess, subphrenic, diagnosis from, 813.
,, air, composition of, in, 794.
,, air-containing viscera, rupture of, causing, 774.
,, amphoric breathing in, 787.
,, bell sound in, 787.
,, bruit de fistule in, 789.
,, causes of, 768.
,, ,, emphysema, 769.
,, ,, empyema, 768.
,, ,, injury, 772.
,, ,, phthisis, 768.
,, ,, respiratory efforts, violent, 770.
,, ,, rupture of air-containing viscera other than lungs, 774.
,, cavity in lung, diagnosis from, 811.
,, chest wall, rare after punctured wounds of, 772.
,, chest wall, rare after fractured rib, 773.
,, chest wall, not always following open wounds of, 773.
,, closed, 806.
,, collapse in, 781.
,, consequences, physiological, of, 777.
,, death, causes of, in, 797.
,, decubitus in, 783.
,, diagnosis of, 810.
,, ,, from cavity in lung, 811.
,, ,, rupture of diaphragm, 812.
,, ,, stomach resonance, 811.
,, ,, subcutaneous emphysema, 812.
,, ,, subphrenic abscess, 813.
,, diaphragm, rupture of, diagnosis of, from, 812.
,, displacement of organs in, 778, 784.
,, ,, diminution of respiratory capacity from, 779.
,, ,, effect of, on opposite lung, 778.
,, ,, explanation of, 778.
,, double, 809.
,, dulness, much fluid in pleura without, in, 786.
,, duration of, 795.
,, effusion in, 793.
,, ,, purulent, 805.
,, ,, serous, 802.
,, ,, without any, 800.
,, emphysema as cause of, 768.
,, ,, subcutaneous, diagnosis from, 812.

Pneumothorax, empyema as cause of, 768.
,, etiology of, 768.
,, fistule, bruit de, in, 789.
,, fluid, much, without dulness, 786.
,, forms of, 806.
,, ,, closed, 806.
,, ,, double, 809.
,, ,, healthy, in apparently, 808.
,, ,, latent, 807.
,, ,, open, 806.
,, ,, partial, 806.
,, ,, recurrent, 809.
,, ,, valvular, 806.
,, fractured rib, rare after, 773.
,, healthy, in apparently, 808.
,, heart, displacement of, in, 784.
,, history of, 767.
,, hydro, 816.
,, incision, by free, treatment of, 818, 821.
,, injury of chest walls, not always causing, 773.
,, ,, of lungs, not always causing, 774.
,, latent, 507, 807.
,, liver, displacement of, in, 784.
,, lung, laceration of, not always causing, 774.
,, mechanism of, 774.
,, metallic tinkling in, 789.
,, mortality of, 795.
,, onset of, 780.
,, open, 806.
,, pain in, 781.
,, paracentesis, treatment of, by, 818.
,, ,, after, 695.
,, partial, 781, 806.
,, pathology, 789.
,, perforation in lung, seat of, 791.
,, phthisis in relation to, 421, 506, 768.
,, pleural tension in, 629.
,, pneumonia and, 291.
,, prognosis of, 798.
,, ,, in respect of danger to life, 798.
,, ,, in respect of duration of life, 799.
,, ,, in respect of recovery, 799.
,, pulse in, 782.
,, punctured wound of chest wall, rare after, 772.
,, pyo-, 817.
,, recovery from, without effusion, 800.
,, ,, with purulent, 805.
,, ,, with serous, 802.
,, recurrent, 809.
,, respiratory capacity, diminution of, from, 779.
Pneumothorax, respiratory effort, producing, 770.
,, rib, fractured, rarely causing, 773.
,, rupture of diaphragm, diagnosis from, 812.
,, rupture of other air-containing viscera causing, 774.
,, signs, physical, of, 783.
,, stomach resonance, diagnosis from, 811.
,, subphrenic abscess, diagnosis from, 813.
,, succussion in, 788.
,, symptoms of, 780.
,, temperature in, 782.
,, tension, intrapleural, in, 629.
,, third attack of, 801.
,, treatment of, 814.
,, ,, early stage of, 814.
,, ,, later stages of, 816.
,, ,, hydro-, 816.
,, ,, pyo-, 817.
,, ,, by incision, 818, 821.
,, ,, paracentesis, 818.
,, tuberculin treatment in association with subsequent, 803.
,, valvular, 806.
,, viscera, rupture of other air-containing, causing, 774.
,, wounds of chest wall not necessarily followed by, 772, 773.
,, wounds of lung not necessarily followed by, 774.
Polymorphic pleurisy, 717.
Poly-orrhomenitis, 749.
Polypoid tubercular vegetations, 77.
Poly-serositis, 749.
Population, density of, in relation to phthisis, 441.
Post-tracheotomic vegetations, 96.
Potters' pneumo-koniosis, 198.
Pouches, œsophageal, 100.
,, tracheal, 100.
Poultice, how to make, 140.
Poumon, insuffisance aiguë du, 779.
Pregnancy, bronchitis in, 183.
,, phthisis and, 579.
,, pneumonia and, 298.
Protozoan infection of lung, 859.
Prune-juice sputum, 380.
,, ,, in pneumonia, 274.
Pseudo-tubercle bacillus, 433.
Pseudo-tuberculosis hominis, coccidioidal, 859.
,, ,, streptotricha, 854.
Psoas abscess, empyema resembling, 708.
Pterygoid chest, 453.
Pulmonary artery, aneurysm of, 382.
,, ,, ,, clotting in, 384.
,, ,, ,, rupture of, 384.
,, ,, blood pressure in, 239.
,, embolism, 254.
,, ,, air, 359.
,, ,, cardiac in origin, 355.
,, ,, diagnosis of, 360.

Pulmonary embolism, fat, 358.
,, ,, forms of, 360.
,, ,, prognosis of, 357.
,, ,, treatment of, 360.
,, ,, venous in origin, 355.
,, infarct, 351.
,, ,, causes of, 351.
,, ,, pathology of, 351.
,, ,, signs, physical, of, 353.
,, ,, symptoms of, 353.
,, osteo-arthropathy, 493.
,, thrombosis, 361.
,, ,, symptoms of, 362.
,, ,, treatment of, 362.
,, vein, thrombosis of, 238.
,, vessels, aneurysm of, 382.
,, ,, erosion of, 386.
,, ,, ulceration of, 386.
Pulsating empyema, 720.
,, ,, diagnosis of, 722.
,, ,, explanation of, 723.
,, ,, forms of, 721.
,, ,, not necessarily fatal, 724.
Pulsation, extra-pleural, 721.
,, intra-pleural, 721.
Pulse in phthisis, 481.
Pulse-rate in pneumonia, 280.
,, ,, slow, 280.
Pulse-respiration ratio in pneumonia, 271.
,, ,, during convalescence, 272.
Putrid bronchitis, 163.
,, ,, diagnosis of, 166.
,, ,, history of, 167.
,, ,, sputum in, 163.
,, ,, treatment of, 167.
Pyæmia in pneumonia, 292.
Pyæmic form of actinomycosis, 849.
Pyo-pneumothorax, recovery, cases of, 805.
Pyo-pneumothorax subphrenicus, 774.

Quinine in whooping-cough, 615.

Race in relation to phthisis, 439.
Râles, 15.
Rash after operation for empyema, 733.
Recurrent empyema, 719.
Redux friction, 667.
Relaxed lung, 222.
Renal disease in relation to bronchitis, 127, 183.
,, ,, phthisis, 520.
,, ,, pneumonia, 285, 294.
Respiration, rate of, in pneumonia, 270.
,, typus inversus of, in emphysema, 209.
Respiratory croaking in babies, 62.
,, neuroses, 566.
,, obstruction, 19.
,, ,, causes of, 22.
,, ,, diagnosis of, 22.
,, ,, prognosis of, 22.
,, ,, results of, 21.
,, ,, symptoms of, 20.
,, organs, malformation of, 18.
,, oscillation, 636.
Rheumatic fever and bronchitis, 183.
Rheumatic fever and phthisis, 520.
,, ,, pleurisy, 651.
Ribs, fractured, rarely causing pneumothorax, 773.
Riviera, The, for the phthisical, 559.
Root cancer of the lung, primary, 103, 882.
Root of lung, dilated left auricle pressing on, 108.
,, pericardial effusion pressing on, 107.
,, primary tumour of, 103.
Rupture of lung, experiments on, 223, 771.
,, in whooping-cough, 605.

Sarcoma of lung, 875.
,, primary, 877.
Scarlet fever, laryngitis in, 37.
,, pneumonia in, 295.
Season, influence of, on bronchitis, 109.
Senile emphysema, 218.
Septic bronchitis, 163.
Septic pneumonia, 323.
Septicæmia after empyema, 709.
Serous effusion, pleuritic, result of tubercle, 48.
Serous expectoration, 162, 691.
Serous membranes, general tuberculosis of, 749.
Siderosis, 197.
Signe de cordeau, 661.
Singers' nodes on vocal cords, 34.
Skodaic resonance, 222.
,, ,, in pleuritic effusion, 665.
,, ,, pneumonia, 277.
Sleep-sweats in phthisis, 466.
Small-pox, laryngitis in, 37.
Sneezing, paroxysmal, 828.
Spasm of the diaphragm, 56.
,, larynx, 56.
,, ,, in the adult, 60.
,, ,, in the child, 58.
Spasmodic croup, 35.
,, laryngitis, 35.
Spine, cervical, disease of, causing pressure on trachea, 101.
Spirals, Curschmann's, 160.
Spleen, empyema perforating into, 707.
,, in phthisis, 426.
,, in pneumonia, 258.
Sprays in bronchitis, 135.
Sputa, cocta, 124.
,, cruda, 124.
,, margaritacea, 166.
,, muco-purulenta, 124.
,, pearly, 166.
,, pituitosa, 125.
Sputum in bronchiectasis, 190.
,, bronchitis, 124.
,, phthisis, 462.
,, pneumonia, 274.
,, putrid bronchitis, 163.
See also *expectoration*, *hæmoptysis*, etc.
Staphylococcus empyema, 715.
Stomach, empyema bursting into, 707.
,, resonance, diagnosis of pneumothorax from, 811.
,, tubercular ulceration of, in phthisis, 423.

Stramonium in asthma, 591.
Streptococcus empyema, 714.
Streptothrix Israeli, 854.
,, pseudo-tuberculosis hominis, 854.
Stridor, bronchial glands, enlarged, causing, 165.
,, congenital infantile, 62.
Subclavian murmur in phthisis, 482.
Subphrenic abscess, empyema resembling, 718.
,, ,, pneumothorax resembling, 813.
,, pyo-pneumothorax, 774.
Succussion in pneumothorax, 788.
,, ,, causes of absence of, 788.
Suffocative catarrh, 150, 324, 325.
Suppuration in mediastinum, 106.
,, of bronchial glands, 105.
Surgical operations for phthisis, 545.
Sweats, night or sleep, in phthisis, 460.
,, ,, ,, treatment of, 546.
Symphysis pleuræ, 753.
,, ,, pathology of, 754.
,, ,, prognosis of, 755.
,, ,, results of, 755.
,, ,, symptoms of, 755.
,, ,, treatment of, 755.
Syphilis of bronchi, 89.
,, larynx, 84.
,, ,, cicatrization in, 85, 88.
,, ,, in children, 85.
,, lung, 836.
,, pleura, 836.
,, trachea, 89.
,, relation of, to bronchitis, 183.
,, ,, phthisis, 516.
,, ,, pleurisy, 651.
Syphilitic fibrosis of lung, 838.
,, phthisis, 839.
,, pneumonia, 836.

Tasmania for the phthisical, 561.
Tension, intrapleural, 625.
,, ,, in empyema, 636.
,, ,, pneumothorax, 629.
,, ,, serous effusion, 633.
,, ,, respiratory oscillation in, 636, 637.
Thrombosis of femoral vein in phthisis, 512.
,, pulmonary vessels, 238, 361.
,, heart in empyema, 709.
,, ,, pneumonia, 292.
,, in phthisis, 505.
Thyroid gland compressing trachea, 97.
Tongue, tubercular ulceration of, in phthisis, 425.
Torquay for the phthisical, 563.
Trachea, anatomy of, 4.
,, compression of, by bronchocele, 97.
,, ,, exophthalmic goître, 99.
,, ,, new-growths of thyroid, 101.
,, ,, other affections of thyroid, 99.
,, hernia of,
,, pressure on, by aneurysm, 102.
Trachea, pressure on, by disease of œsophagus, 101.
,, ,, ,, spine, 101.
,, ,, goître, 97.
,, ,, mediastinal tumour, 102.
,, syphilis of, 86, 89.
,, tumours of, malignant, 96.
,, ,, non-malignant, 95.
,, tubercle of, 83.
Tracheal pouches, 100.
Tracheitis, chronic, 156.
Tracheo-bronchitis of adult, 147.
,, ,, ,, chronic, 156.
,, ,, ,, complications of, 148.
,, ,, ,, duration of, 148.
,, ,, ,, prognosis, 148.
,, ,, ,, treatment, 149.
Tracheocele, 99.
Tracheotomy for diphtheritic laryngitis, results of, 49.
,, foreign body, 30.
,, vegetations after, 96.
Traumatic delirium, 305.
Tubercle of bronchi, 83.
,, larynx, 74.
,, trachea, 83.
,, in relation to empyema, 749.
,, ,, pleurisy, 652.
Tubercle-bacillus, changes produced by, in general, 402.
,, in lung, 405.
,, ,, cultivation of, 402.
,, ,, history of, outside body, 427.
,, ,, in man and cattle, not the same, 432.
,, ,, pseudo, 433.
,, ,, staining of, 402.
Tubercular bronchial glands, 104.
,, empyema, 716.
,, infection, conditions influencing, 438.
,, ,, from cattle, 432.
,, ,, ,, flesh, 432.
,, ,, ,, man to man, 435.
,, ,, ,, among attendants, 437.
,, ,, ,, in institutions, 437.
,, ,, ,, milk, 431.
,, ,, modes of, feeding, 434.
,, ,, ,, inhalation, 435.
,, ,, ,, inoculation, 434.
,, infiltration, 408.
,, meningitis in phthisis, 510.
,, pleurisy, 746.
,, ,, in phthisis, 420.
,, ulceration of bucco-pharynx in phthisis, 424.
,, ,, duodenum in phthisis, 423.
,, ,, intestines in phthisis, 421.
,, ,, œsophagus in phthisis, 423.

Tubercular ulceration of peritoneum in phthisis, 510.
,, ,, stomach in phthisis, 423.
Tubercular vegetations, polypoid, in larynx, 77.
Tuberculin treatment of phthisis, 540.
,, ,, ,, occurrence of pneumothorax during, 803.
Tuberculosis, acute, of lung, 496, 510.
,, general, in phthisis, 510.
,, ,, of serous membrane, 749.
,, in cattle, 431.
,, children, 429.
,, fœtus, 451.
Tumour, air-containing, in neck, 100.
,, of lung and pleura, 872.
,, of mediastinum, 102.
,, ,, diagnosis of in general, 102.
,, ,, ,, from aneurysm, 103.
,, ,, pressing on trachea, 102.
,, of neck compressing trachea, 101.
,, of œsophagus pressing air-tubes, 107.
,, primary, at root of lung, 103.
Turtle lung, 187.
Typhoid bacillus and empyema, 716.
,, ,, pneumonia, 295.
Typhoid fever, laryngitis in, 37.
Typhoid fever and phthisis, 518.
,, ,, pneumonia, 295.
Tympanites in pneumonia, 287.
Typus inversus of respiration in emphysema, 209.

Ulceration of bowels, tubercular, in phthisis. 510.
,, pulmonary vessels, 386.
Urea in urine in pneumonia, 284.

Vaccination in relation to phthisis, 517.
Vacuoles pulmonaires, 327.
Valve-shock in phthisis, 482.
Vein, pulmonary, thrombosis of, 238.
Vena cava, kinking of, cause of death in pleuritic effusion, 663.
Venesection in bronchitis, 141.
,, pneumonia, 308.
Venous congestion causing bronchitis, 111.
,, ,, effect on heart, causing dilatation, 129.
,, ,, ,, causing murmurs, apex, 130.
,, ,, ,, causing murmurs, tricuspid, 130.
,, ,, effect on lungs, 129.
Ventnor for the phthisical, 563.
Vessels perforated by empyema, 707.
Vicarious emphysema, 220.
,, menstruation, 239.
,, ,, relation of to hæmoptysis, 380.
Vital capacity, reduction of, in emphysema, 208.
,, ,, pleuritic effusion, 208.
Vocal cords, cadaveric position of, 65.
,, median position of, 65.
Vomiting in phthisis, 483.
,, ,, treatment of, 549.
,, pneumonia, 283.

Washing out empyema, 731.
Water cures for bronchitis, 145.
Weak heart, congestion of lungs in, 235.
Webs of larynx and trachea, acquired, 88.
,, ,, ,, congenital, 88.
Whooping-cough, 600.
,, complications of, 603.
,, ,, associated with, in catarrh, 603.
,, ,, ,, cough, 604.
,, ,, ,, nervous system, 605.
,, convulsions in, 606.
,, diagnosis of, 610.
,, duration of, 603.
,, etiology of, 608.
,, frenal ulcer in, 605.
,, hæmorrhage in, 604.
,, hemiplegia in, 606.
,, history of, 600.
,, measles, relation of, to, 609.
,, mortality of, 611.
,, pathology of, 607.
,, prognosis of, 610.
,, rupture of lung in, 605.
,, sequelæ of, 606.
,, stages of, catarrhal, 600.
,, ,, convalescent, 603.
,, ,, convulsive, 601.
,, symptoms of, 600.
,, theories of, 607.
,, treatment of, 612.
,, ,, paroxysm, 613.
,, ulcer, frenal, in, 605.
Winter resorts for the phthisical, 558.
,, ,, Africa, Southern, 561.
,, ,, Algiers, 560.
,, ,, Alps, 558.
,, ,, America, Western, 561.
,, ,, ,, South, 562.
,, ,, Australia, 561.
,, ,, Canary Islands, 560.
,, ,, Egypt, 560.
,, ,, Home, 563.
,, ,, Madeira, 560.
,, ,, New Zealand, 561.
,, ,, Riviera, 559.
,, ,, Tasmania, 561.

Vol. I. comprises pages 1–398. Vol. II. pages 399 to the end.

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INDEX TO AUTHORS.

PAGE

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ANDERSON (Prof. M'Call), Skin Diseases, 13
BLYTH (A. W.), Foods and Poisons, . . 31
BURNET (R., M.D.), Foods and Dietaries, 33
BURY (Judson, M.D.) Clinical Medicine, . 7
CAIRD & CATHCART, Surgical Handbook, 22
CLARK (Sir Andrew), Fibroid Phthisis, . 9
CRIMP (W. S.), Sewage Disposal Works, 21
DAVIES (Lt-Col.), Hygiene, . . . 23
DAVIS (Prof. J. R. A.), Biology, . . 30
——The Flowering Plant, ... 30
——Zoological Pocket-book, ... 30
DONALD (Arch., M.D.), Midwifery, . . 25
DONKIN (H. Bryan), Diseases of Childhood, 9
DUCKWORTH (Sir D., M.D.), Gout, . 10
DUPRÉ and HAKE, Manual of Chemistry, 32
ELBORNE (W.), Pharmacy, ... 32
ELDER and FOWLER, Diseases of Children, ... 24
FOXWELL (A., M.D.), Heart and Lung Diseases, ... 18
GARROD (A. E., M.D.), Rheumatism, . 11
HADDON (Prof.), Embryology, . . . 18
HELLIER (Dr.), Infancy and Infant-Rearing, 35
HILL (Dr.), Physiologist's Note-Book, . 27
HORSLEY (V.), Brain and Spinal Cord, . 15
HUMPHRY (G., M.D.), Manual of Nursing, 33
HUNTER (Wm., M.D.), Pernicious Anæmia, ... 19
JAKSCH (Prof. R. v.), Clinical Diagnosis, 8
LAFAR (Dr. Franz), Technical Mycology, 26
LANDIS (Dr.), Management of Labour, . 25
LANDOIS and STIRLING'S Physiology, 5
LEWIS (Bevan), Mental Diseases, . . 16
MACALISTER (Prof.), Human Anatomy, 4
MACREADY (J., F.R.C.S.), Ruptures, . 19
MANN (Prof. Dixon, M.D.), Forensic Medicine and Toxicology, ... 17
MERCIER (Ch.), Asylum Management, . 17
MEYER and FERGUS, Ophthalmology, . 14
OBERSTEINER and HILL, Nervous Organs, ... 18
OPPENHEIMER (Dr. C.), Ferments, . 27
PAGE (H. W., F.R.C.S.), Railway Injuries, 20
PAWLOW (Prof.), Digestive Glands, . 13
PHILLIPS (Dr. J.), Diseases of Women, . 25
PORTER and GODWIN, Surgeon's Pocket-book, ... 23
REID (Geo., D.P.H.), Practical Sanitation, 34
RICHMOND (H. Droop), F.C.S., Dairy Chemistry, ... 26
RIDDELL (J. Scott, M.D.), Ambulance, . 35
RIEDER and DELÉPINE, Urinary Sediments, ... 6
SANSOM (A. E., M.D.), Diseases of the Heart, 12
SEXTON (Prof.), Quantitative Analysis, . 32
——Qualitative Analysis, ... 32
SMITH (Johnson, F.R.C.S.), Shipmasters' Medical Guide, ... 33
SQUIRE, (Ed. J., M.D.), Consumption, 34
STIRLING (Prof.), Practical Physiology, . 28
——Practical Histology, ... 29
THOINOT, MASSELIN, and SYMMERS, Bacteriology, ... 24
THORBURN (W.), Surgery of Spine, . 20
THORNTON (J.), Surgery of Kidneys, . 20
WEST (S., M.D.), Diseases of the Organs of Respiration, ... 12
WESTLAND (A., M.D.), Wife and Mother, 35
WOOD (F.), Sanitary Engineering, . . 21
SCIENTIFIC SOCIETIES (Year-book of), 36

INDEX TO SUBJECTS.

PAGE

AMBULANCE, ... 35
ANATOMY, Human, ... 4
Anatomy and Physiology (*Journal of*), . . 15
ASYLUM MANAGEMENT, . . . 17
BACTERIOLOGY, ... 24, 27
BIOLOGY, ... 30
BOTANY, ... 30
BRAIN, The, ... 15, 16, 17, 18, 19
CHEMISTRY, ... 32
CHILDHOOD, Diseases of, . . . 9, 24
CLINICAL Diagnosis, ... 8
CLINICAL Medicine, ... 7
CONSUMPTION, ... 9, 12, 18, 34
DAIRY CHEMISTRY, ... 26
DIETARIES for the Sick, ... 33
DIGESTION, ... 13
EMBRYOLOGY, ... 18
EYE, Diseases of the, ... 14
FERMENTATION, ... 27
FOODS, ... 31, 33
FORENSIC MEDICINE, ... 17
GOUT, ... 10
HEART, Diseases of the, ... 12
HISTOLOGY, ... 29
HYGIENE and Public Health, . 21, 23, 34
INFANTS, Rearing of, ... 35
INSANITY, ... 16, 17
KIDNEYS, Surgery of the, ... 20
LABORATORY Hand-books, . 28, 29, 32
MENTAL DISEASES, . . 16, 17, 18
MICROBIC Diseases of Animals, . . . 6
MYCOLOGY, ... 26
NERVOUS ORGANS, Central, . . . 18
NURSING, Medical and Surgical, . 33, 35
OBSTETRICS, ... 25
PERNICIOUS ANÆMIA, ... 19
PHARMACY, ... 32
PHTHISIS, ... 9, 12, 18, 34
PHYSIOLOGIST'S Note-book, . . . 27
PHYSIOLOGY, ... 5, 13, 28
POISONS, Detection of, . . . 17, 31
RAILWAY INJURIES 20
REFERENCE POCKET-BOOKS—
——Hygiene, ... 23
——Medical, ... 22
——Surgical, ... 22, 23
——Zoological, ... 30
RESPIRATORY DISEASES, . . . 12
RHEUMATISM, ... 11
RUPTURES, ... 19
SANITATION, ... 21, 23, 34
SEWAGE Disposal Works, ... 21
SHIPMASTERS' Medical Guide, . . 33
SKIN, Diseases of the, ... 13
SPINAL Cord, ... 20
SURGERY of Brain, ... 15
——of Kidneys, ... 20
——Military, ... 23
——Minor, ... 22
——Ruptures, ... 19
——of Spinal Cord, ... 20
URINARY SEDIMENTS, Atlas of, . . 6
VETERINARY MEDICINE, . . 6, 24
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		PAGE
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Fibroid Phthisis,	SIR ANDREW CLARK, M.D.,	9
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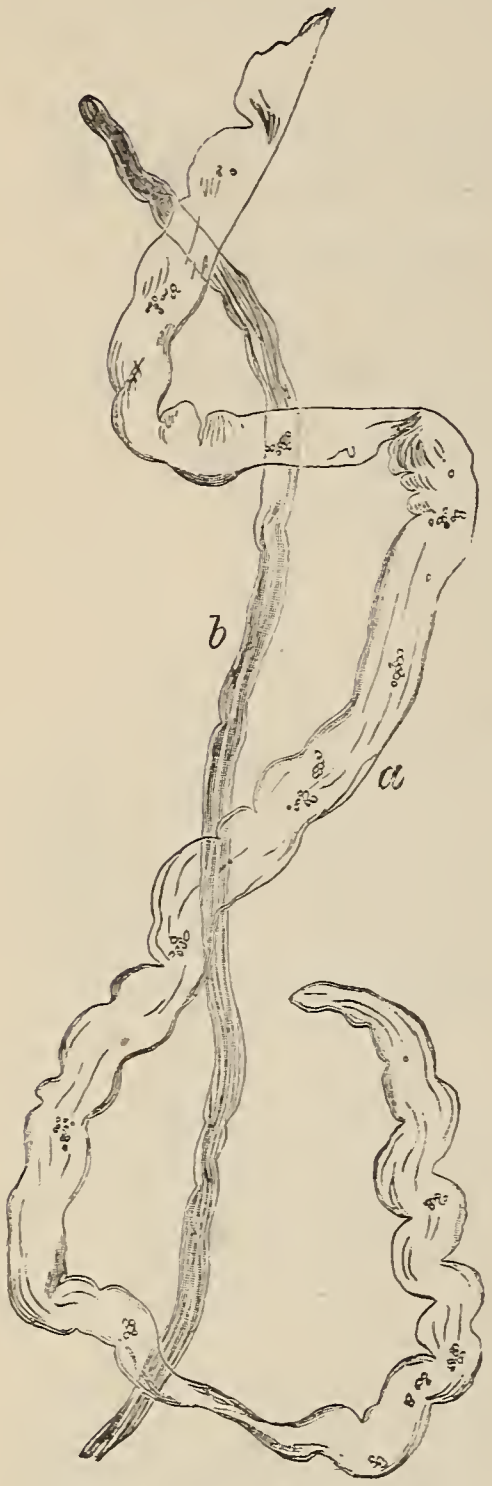

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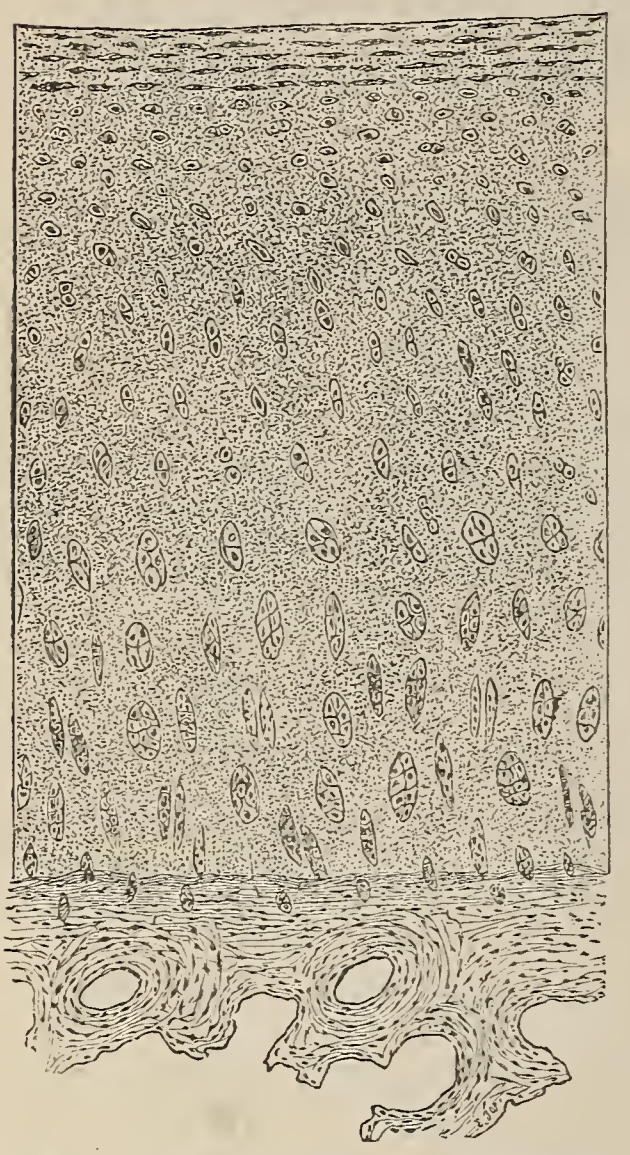

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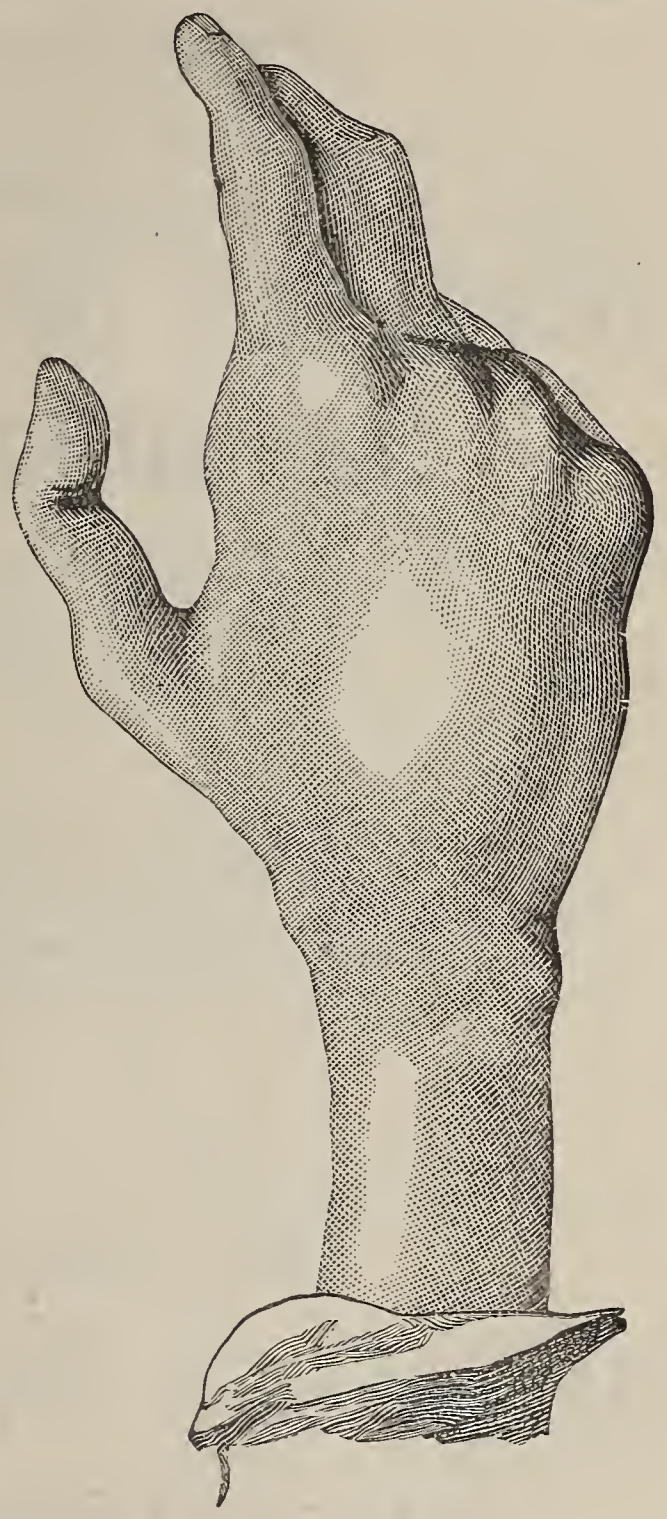

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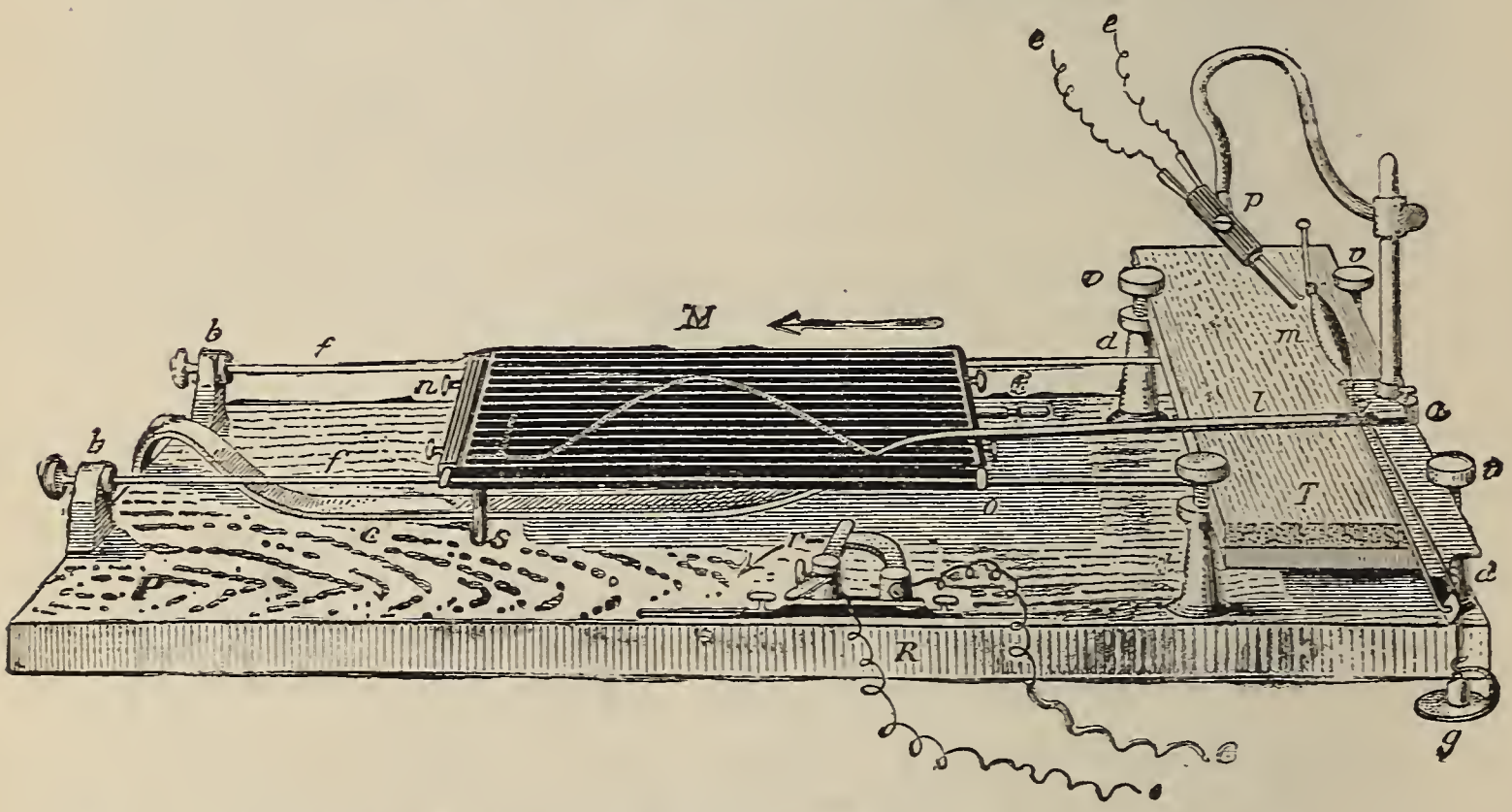

Fig. 118.—Horizontal Myograph of Frédéricq. *M*, Glass plate, moving on the guides *f*, *f*; *l*, Lever; *m*, Muscle; *p*, *e*, *e*, Electrodes; *T*, Cork plate; *a*, Counterpoise to lever; *R*, Key in primary circuit.

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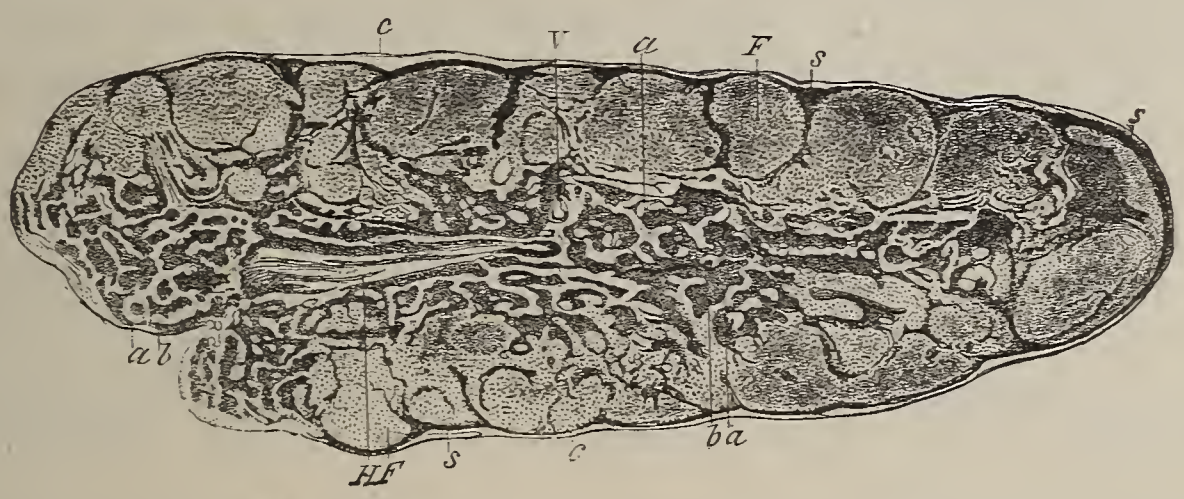

Fig. 200.—L.S., Cervical Ganglion of Dog. *c*, Capsule; *s*, Lymph sinus; *F*, Follicle; *a*, Medullary cord; *b*, Lymph paths of the medulla; *V*, Section of a blood-vessel; *HF*, Fibrous part of the hilum, × 10.

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